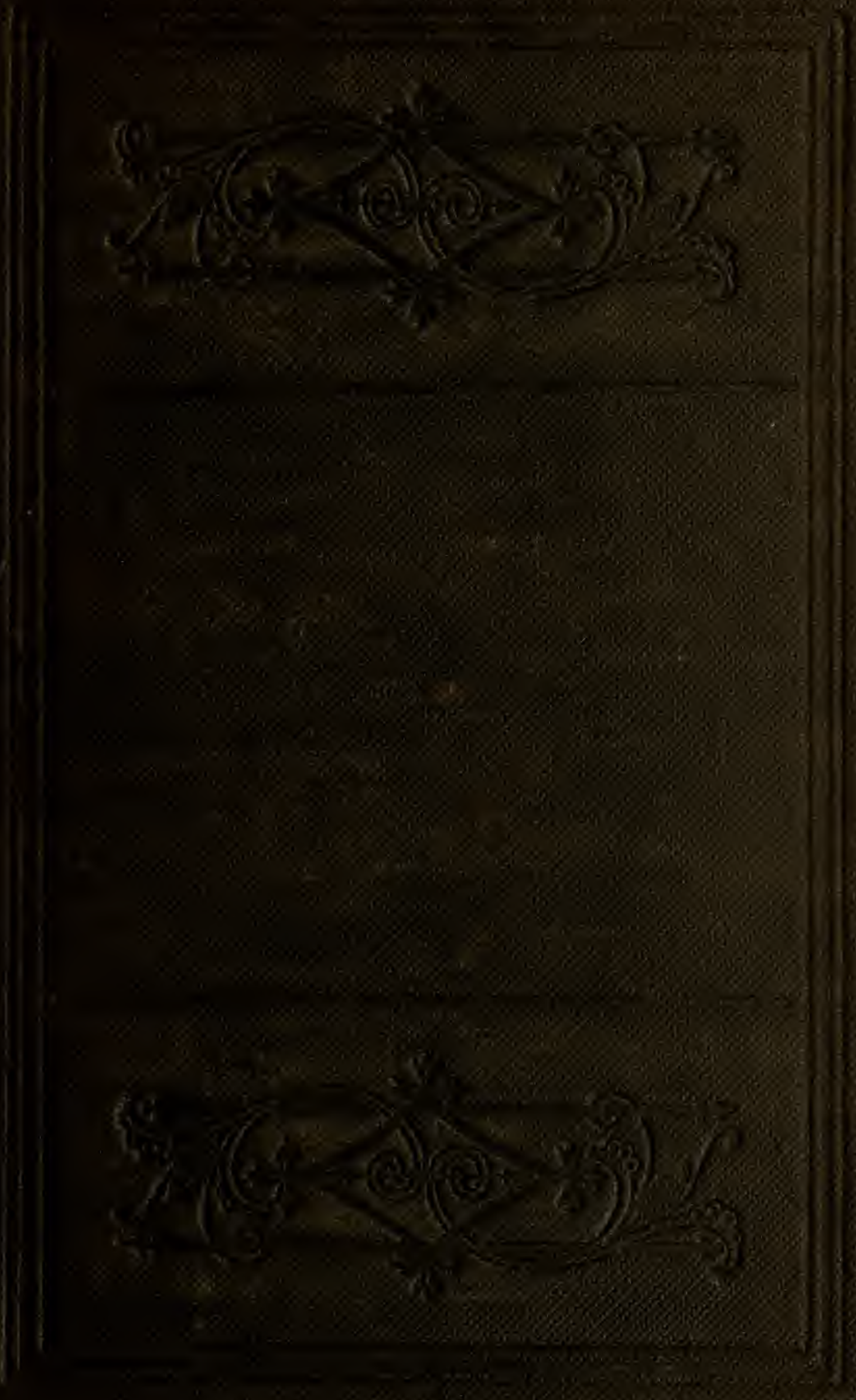

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1875

MANUAL

OF

PATHOLOGICAL ANATOMY

BY

C. HANDFIELD JONES, M.B. CANTAB., F.R.S., F.R.C.P.

PHYSICIAN TO AND LECTURER ON CLINICAL MEDICINE AT ST. MARY'S HOSPITAL

AND

EDWARD H. SIEVEKING, M.D., F.R.C.P.

PHYSICIAN TO ST. MARY'S AND THE LOCK HOSPITALS, PHYSICIAN EXTRAORDINARY TO THE QUEEN, PHYSICIAN IN ORDINARY TO THE PRINCE OF WALES

SECOND EDITION

REVISED, ENLARGED, AND EDITED BY

JOSEPH FRANK PAYNE, M.B. OXON., F.R.C.P.

FELLOW OF MAGDALEN COLLEGE, OXFORD, ASSISTANT PHYSICIAN TO AND LATE DEMONSTRATOR OF MORBID ANATOMY AT ST. THOMAS'S HOSPITAL

LONDON
J. & A. CHURCHILL, NEW BURLINGTON STREET
1875

CATALOGUED.
E. J. C.

1166

PREFATORY NOTICE TO THE FIRST EDITION.

THE Authors of the present work have desired to lay before their professional brethren an outline of what is known in the domain of Pathological Anatomy. The absence of any original work in the English language, which embraces the whole subject, must be their apology for having made the attempt. They have sought to place before the reader a summary of ascertained facts, together with the opinions of the most eminent pathologists of this and other countries. They have regarded it as their duty to select, as far as possible, the best fruits from the harvest gathered by other labourers in this wide and interesting field. At the same time they have sought not to speak solely on the faith of others, even the highest authorities, but to investigate, as much as possible, for themselves, the correctness of the statements they adopted. They felt that, in some instances, better illustrations might have been obtained by borrowing from other works, but they were of opinion that the present manual would bear a stamp of greater truthfulness if the drawings were taken from objects seen and examined by themselves. They have therefore preferred (with few exceptions only) to use such illustrations as their own portfolios supplied. Although small drawings, in black and white, necessarily fail to give the important elements of size and colour, almost essential to illustrations of Pathological Anatomy, the Authors hope that the masterly treatment of Mr. Bagg has achieved as much as could be done by wood engraving. They have divided the subject in the manner indicated in the Table of Contents, and are each individually responsible for the chapters which they have treated.

They conclude by expressing a hope that the vast extent of the subject, and of the material they had to deal with, will serve, in some measure, as an apology for the deficiencies which they are fully conscious of, and for which they ask the kind and lenient consideration of the Medical Profession.

C. HANDFIELD JONES.
EDWARD H. SIEVEKING.

London, *August*, 1854.

PREFACE TO SECOND EDITION.

We have much pleasure in stating that in preparing the Second Edition of this work Dr. Payne has been by far the chief agent; and that, although we have supervised and carefully examined his labours, the additions and improvements necessary to bring it up to the mark of the present day are entirely his work. We think he has performed his part with great ability, and we trust that the volume now affords to the student a comprehensive summary of all that is important in the science of Pathological Anatomy.

C. HANDFIELD JONES.
EDWARD H. SIEVEKING.

December, 1874.

In the preparation of the Second Edition the following textbooks have been constantly consulted, but it has not been thought necessary as a rule to refer to special passages of these works:—

Rokitansky, "Lehrbuch der Pathologischen Anatomie:" Third Edition, Vienna, 1855-61. Förster, "Handbuch der Pathologischen Anatomie:" Second Edition, Leipzig. 1863-65. Cornil and

Ranvier, "Manuel d'Histologie Pathologique:" First and second parts, Paris, 1869-73. Rindfleisch, "Handbuch der Pathologischen Gewebelehre:" First Edition, Leipzig, 1867-69.

References have generally been given to the works of other authors quoted, but indulgence must be asked for the numerous unavoidable omissions.

The new illustrations introduced have been, for the most part, drawn on the wood by Mr. Wesley, either from original preparations by the Editor, or from other sources, which have been duly indicated. We have to thank the Council of the Pathological Society of London for permission to copy some figures from plates in the Transactions of that Society, and for the loan of wood blocks. Similar acknowledgments are due to Dr. Caton, Dr. Lockhart Clarke, Dr. Cobbold, and Dr. Dickinson. For three drawings we are indebted to Mr. Henry Arnott.

J. F. PAYNE.

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MANUAL

OF

PATHOLOGICAL ANATOMY.

PART I.

GENERAL PATHOLOGICAL ANATOMY.

CHAPTER I.

GENERAL OBSERVATIONS.

THE object of General Pathology is to examine the various morbid processes which may occur in the human body, and to obtain, thereby, such an insight into their nature, that they may not be looked on as unknown entities, but that being comprehended as far as is possible themselves, the various effects they produce, the particular instances of their action, may be understood also.

The course we propose to follow is to describe briefly, (I.) The morbid alterations of the several great functions. (II.) Those of the blood. (III.) Those of the various tissues, considered generally. (IV.) The superadded formations or growths, the so-called tumours. (V.) Parasitic beings, whether animal or vegetable.

Some general observations must, however, be premised. The term *Morbid Anatomy* hardly needs explanation; as ordinary anatomy implies the study of, and acquaintance with, the healthy structure, so morbid anatomy implies the same of diseased structure. The meaning of *Pathology* may be clearly conceived, by considering that of its twin sister, Physiology; as the latter imports the knowledge of the natural actions of healthy organs, so does the former that of the unnatural actions of diseased or disturbed organs. Physiology has her vital stimuli; Pathology her stimuli or excitants to unhealthy life. This expression brings us to notice a point which has been excellently illustrated by Professor Simon. He remarks that many unnatural or diseased conditions are not really unnatural in themselves, but are the proper and necessary consequences of some cause or influence which has acted upon a healthy body. The state of skin which a severe burn produces is, certainly, very unnatural and diseased, but the inflammatory and exudative processes which have produced it are quite natural, under the circumstances that have occurred; they are the proper reaction of a healthy organism to the unnatural stimulus of extreme heat, and are called forth in the same way as is the healthy flow of blood into a chilled part by the action of kindly warmth. So in a case of variola, the skin covered all over with unsightly pustules is in a very unnatural state; but it is not the eruption, nor the constitutional disturbance, neither, that is really unnatural, but the presence of a certain quantity of infectious matter in the blood, which, acting on a perfectly natural system, thus calls forth its expulsive efforts. If we slightly alter this perfectly natural state, as by premising vaccination, then the introduction of the variolous poison no longer produces the same morbid effects, and we say the system is protected. The fact is, the system is changed from its originally perfectly natural condition, and will no longer respond to the unnatural stimulus. We find, it may be, a portion of the brain so soft as to resemble cream, quite broken up and disorganized; but we look further and find that the artery supplying it with blood has been plugged up or tied, and we then see that the *locus* of the disease was not really in the brain, but in the artery; it would have been abnormal had the brain, deprived of its supply of blood, retained its natural texture. Or, again, we see a person suffering from violent epileptic convulsions, but he passes a large worm from the bowels, and the attacks cease; in the case of this individual, the convulsions were the natural expression of the unnatural irritation to which the brain was subjected. Many like instances might be mentioned, and they certainly show that disease is, very often, not to be regarded as a special entity of a peculiar, strange kind, but as the natural result of the endowments and qualities belonging to our bodily organs, when those organs are acted on by certain unnatural stimuli. Hence we can better understand that many diseases have a regular and normal course, made up, so to speak, of a succession of necessary results, which, however, is liable to be disturbed by various extrinsic causes. For instance, a person has ague, the paroxysms occurring in regular succession; he takes quinine, and they diminish and disappear, the course of morbid action is interfered with and broken. Or, a child has hooping-cough, and the disease is proceeding in its usual course, but in consequence of exposure to cold, he is attacked with inflammation of the lungs, and the paroxysms characteristic of the disease are, to a great degree, interrupted; the hooping-cough is merged in the pneumonia. Or, again, a person has tubercular deposit in his lungs; the natural tendency of this is to soften, break down, and be expectorated, together with the involved tissue, while, as fresh deposits take place, more and more of the organ is destroyed; but, before this can happen, inflammation is set up to such an extent, in the surrounding tissue, that life is cut short, not by the effects of the tubercular destruction directly, but by the intercurrent inflammation.

The unnatural stimuli, provoking the succession of morbid actions, are often termed the *Exciting* causes of a disease; they may be adequate, when powerful of themselves to produce their effect, or may need the assistance of other causes, generally of a debilitating nature, which are called *Predisposing.*

But the question now occurs, whether all diseases are of the kind above mentioned, whether all can be regarded as the natural results of certain foreign injurious influences operating on the system. To this the answer, in the present state of pathology, must be, I think, Certainly not. There are very many cases where we cannot point out any exciting cause of the existing malady, where it seems to have originated spontaneously, so far as we are able to judge. Of this kind are many instances of decay and degeneration of tissues, very many of mal-assimilation, or mal-secretion, hereditary diseases, and some congenital mal-formations. It may be that, as we advance in knowledge, we shall be able to include more and more of the latter class under the former; that as we obtain more acquaintance with the imponderable influences which are constantly in operation, we shall be able to refer to them as the causes of changes which now appear spontaneous, but from this we are far at present, and must thoroughly recognize the two classes of disease which we have just described. These classes, however, are not (and natural groups never are) rigorously defined; there are numerous instances of an intermediate kind, such as those where a slight exciting cause calls into action an inherited predisposition. We must also notice another great division of diseases into two classes, viz., the Organic and Functional. Of the existence of the latter many of the best pathologists greatly doubt, that is to say, whether it be possible for the mechanism of an organ to be perfectly uninjured, at the time that its function is wrongly performed. Speaking in the strictest sense, and remembering the advances which have been made in detecting morbid alterations formerly unknown, as well as the amount of progress which we may yet look to make, it must certainly be allowed that it is quite possible that the division above mentioned is not founded in reality, and that all diseases are attended with organic change. But, when this is conceded, it remains still perfectly clear that there are not a few diseases, and some very severe, in which no organic alteration whatever can be detected, and it seems further, a point of considerable practical importance that the student should be fully aware of this and alive to it. What are called *idiosyncrasies* are unusual peculiarities of an individual system, in consequence of which it is affected in a different manner by some influences to that which is commonly experienced. Thus some persons are attacked by asthma or bronchitis on inhaling the odour of hay; some are almost poisoned by taking the smallest dose of a mercurial. One at least has been mentioned (by Dr. Prout) who could not eat mutton in any form without being attacked by violent vomiting and diarrhœa. In such persons the qualities and endowments of one or more organs must be essentially different to those of the same parts in the vast majority of mankind. Yet there is not the least reason for supposing that, by any scrutiny, we could detect any structural difference, and they must, therefore, be deemed instances of aberring function.

One remarkable instance it seems worth while to adduce here, which affords an excellent illustration of the connection that may obtain between disordered function and alteration of structure. The Graafian vesicle in the ovary, instinct with a wonderful capacity of life, which only needs its appropriate stimulus to rouse it into that activity which issues in the production of another being, not unfrequently, as if affected by some strange and unnatural stimulus, proceeds to develop itself into a huge anomalous growth, utterly imperfect, and unlike what, under normal conditions, it should have produced, and yet exhibiting some traces such as are found in no other growth of its original destiny, by the formation of several of the natural tissues, skin, teeth, hair, nay, even brain, &c. Here it seems impossible to recognize any other cause of the organic alteration beside the perversion or aberrance of a natural function or endowment. The term *Diathesis* is applied to a certain condition of the general system often inherited, which renders it especially liable to some particular form of disease; thus we speak of the scrofulous or tubercular diathesis, of the gouty diathesis, and so on. If the diathesis or predisposition be strong, a slight exciting cause will be sufficient to induce the malady; if it be absent, no exciting cause may produce any effect. A diathesis may, therefore, be considered as a kind of special weakness. *Degeneration* of a part or tissue implies generally its slow and gradual conversion into some lower kind of structure less fitted for the purpose it has to fulfil, as when cartilage is converted into a kind of fibrous tissue; or it may imply the atrophy and destruction of a part, as of the cortical structure of the kidney in Bright's disease. It is to be regarded in some measure as a local infirmity.

It was formerly a much debated question, whether diseases had their principal seat in the fluids or solids of the body; and each of the two opposed theories has at times been dominant. At the present day we marvel how men could have adopted exclusively one view or the other, and refused to allow to each their share in the production of morbid phenomena. There can be no question, from known physical laws, that the blood must, in very many cases, be the first recipient of aeriform noxious matters, of all such miasmata as those of typhus, smallpox, &c. The instant that these are drawn with the air into the lungs they pass into the blood; for it is impossible that the gases contained in the blood shall not, according to the law of heterogeneous attraction, be exchanged in part for those which are diffused in the air cells and cavities of the lungs. As little doubt can there be that the blood, as it is the first to receive, so it is also the first to be modified and altered from its healthy composition by the inhaled miasm. In the great class of inflammations, the affection of the tissue and of the blood must proceed *pari passu;* so intimately is the blood concerned in every stage of the process, that it may almost be said both blood and tissue are alike the seat of the disease; but the latter manifestly has the initiative. Rheumatic and gouty inflammations must, however, be excepted, in which the blood is apparently the primary seat of morbid alteration. Scrofulous disease has been thought to commence in the blood, and to produce in it a change, of the nature of which we are ignorant, but which issues in the deposit of a peculiar matter in various localities. In diseases arising from excess in eating and drinking, or from unwholesome aliment, the blood in some cases, and the alimentary canal in others, may be primarily affected. When we consider what the processes of nutrition and secretion imply, how the blood is a vast laboratory, in which some secretions are actually prepared, and the materials of others; how it conveys to each part the nutriment that is appropriate to it, and receives back in return principles more or less effete; how continually it is receiving supplies of new matter from without, and undergoing depuration by various appointed emunctories; in short, if we consider how thoroughly the different solid and fluid parts of the frame are correlated, and mutually dependent, we shall perceive most clearly that it is far more important to be fully aware of the extreme liability, nay, necessity, of the solids to be affected by the fluids, and the fluids by the solids, and that thus the disorder of one part may be the exponent of the error of another, than to attempt an almost impossible definition of the exact origin and site of a disease.

MORBID ALTERATIONS OF FUNCTION.

These alterations, which would fill so important a chapter in a work on medicine, must occupy a much less conspicuous place in a work on pathological anatomy. For if such alterations are dependent on any structural changes, whether anatomical or humoral, of the organs, they will be described under another head. If, on the other hand, they cannot be traced to any organic change, but are purely functional derangements, they are then by definition excluded from the scope of this work. The question which has often been raised, whether there is such a thing as purely functional derangement, has already been discussed.

CHAPTER II.

MORBID STATES OF THE BLOOD.

The saying attributed to Cuvier, "Le sang est chair coulant," expresses very fairly the relations that subsist between the nutrient circulating fluid and the solid tissues. As we have already remarked, a change in the former involves almost necessarily a change in the latter, and each of the vital actions of the latter exerts some influence on the former. It is manifest, therefore, that an acquaintance with the healthy properties and morbid alterations of the blood is absolutely essential to a correct study of the phenomena wrought by disease in the solid parts. But the blood itself is a very compound thing, not only as regards the number of different matters it contains, organic and inorganic, not only as regards the manifold additions which it receives from without in the way of nutriment, and those which are poured back into it as the effete residues of tissues, but also as regards its own morphological condition, being made up of solid and of fluid parts, of organized particles floating in an organic liquid. In diseased states of the blood these several parts, or some of their constituents, may be separately affected, and it seems therefore desirable on this account, as well as for the sake of greater precision, to examine first the individual component elements of this fluid with reference to their pathological changes, and afterwards to consider the diseases of the blood taken as a whole. Regarding the blood *per se,* this inquiry would rather belong to special than to general Pathology, but we include it under the latter head on account of the manifest general relations of the blood.

RED AND WHITE CORPUSCLES.

The circulating fluid consists, as we know, of two kinds of organized particles, or corpuscles, floating in a transparent colourless fluid, called the *Plasma*, or *Liquor sanguinis*. The organized particles are the red corpuscles, preponderating immensely in number, and giving to the fluid its characteristic colour, and the white or colourless, which in the healthy state occur somewhat "few and far between," in number only $\frac{1}{500}$th to $\frac{1}{350}$th of the red, and impart to the general mass no distinguishable quality.

Healthy blood has an alkaline reaction, which is that of the *liquor sanguinis*, and is, even in the thinnest layers, quite opaque. The exact proportion of the organized particles to the *liquor* is extremely difficult to determine with accuracy. Most analyses refer only to the *dried* corpuscles, but that of Hoppe, which takes account of the water contained in these particles, is by far the most trustworthy. It is an analysis, not of human blood, but of the venous blood of the horse.

In 1,000 parts of blood are contained—

Corpuscles	326·2
Liquor sanguinis	673·8
	1000

In 1,000 parts of corpuscles—

Water	565
Solids	435
	1000

In 1,000 parts of liquor sanguinis—

Water	908·4
Solids	91·6
	1000

The solids of the liquor sanguinis consist of—

Fibrin	10·1
Albumen	77·6
Fat	1·2
Extractives	4·0
Soluble salts	6·4
Insoluble salts	1·7
	101

Red Corpuscles.—These are round discoid bodies, with two concave surfaces. Their consistence is soft and elastic; they are of a uniform red colour, and are specifically heavier than either the *liquor* or the white corpuscles. They were formerly supposed to be cells, with a distinct envelope and a nucleus; subsequently the possession of a nucleus was denied to them, and they are now regarded as homogeneous masses of colourless stroma, without nucleus or enclosing membrane. Many reagents, by condensing the outermost portion of the globule, produce the appearance of a cell-wall. The colour of these discs is due to the presence in them of a peculiar colouring matter, which is normally quite absent from the liquor, and may be separated from the discs without destroying them, being attached either by mere imbibition, after the manner of a dyed fabric, or else by some easily disturbed chemical affinity. This peculiar distribution of colouring matter in the blood is a matter of great physiological and pathological importance. The optical properties of the blood are, from this cause, very different from those of a solution. Its colour is not entirely derived from transmitted light, like that of a solution, but is partly due to reflection from the surfaces of the globules. A true solution, therefore, would appear darker, though it would be perfectly transparent in thin layers, which blood is not. The same fact explains why blood does not permanently stain the vessels containing it, or diffuse into the neighbouring parts, as for instance bile pigment does; the colouring matter being probably never in actual contact with the walls of the vessels. When the colouring matter leaves the globules, and becomes diffused in the *liquor*, blood has the optical and many of the physical properties of a solution. There are several pathological conditions in which this occurs, but it is worth while to enumerate some of the means by which it can be artificially effected, such as simple dilution with water, freezing and melting again, repeated electric discharges, separation of the gases of the blood; the addition of salts of the bile-acids, of ether, of chloroform, or of alcohol in small quantities. All these agents, it should be remembered at the same time, cause the globules to swell up, and ultimately, to become spherical, while, if continued long enough, they for the most part dissolve the decolorized stroma. It is also worth notice that decomposition of the blood produces the same series of changes if sufficient water be present. In dried blood the form of the corpuscles is preserved for an indefinite time. The chemical character of the blood-colouring matter is not without importance in pathology. The substance attached to the corpuscles has been called *Hœmoglobin* (also *Hœmatocrystallin* or *Cruorin*). It is a body of extremely complex but perfectly definite composition, and may be obtained in crystals. When in solution it readily undergoes decomposition into an albuminous substance called *globulin*, and a coloured substance called *Hœmatin*, which was long taken for the true blood pigment. *Hœmatin* and *Hœmoglobin*, however, differ not only in chemical composition (though both contain iron) and in crystalline form, but are readily distinguished by their optical properties. Each produces peculiar "absorption bands" in the solar spectrum, by which its presence may be certainly recognized. Hæmatin has not been found ready formed in the blood; but its production in a crystalline form combined with HCl, constitutes a valuable test for the presence of blood (Teichmann's *Hœmin* crystals). These crystals are frequently found as a pathological product in old extravasations of blood; but the crystals most commonly found in such situations are composed of a third colouring matter obtained from the blood, and known as *Hœmatoidin*. This body contains no iron, and resembles, if it be not identical with, a body obtained from the bile, called Bilirubin.

Hæmatoidin is produced by the decomposition of hæmatin, and hence (supposing that it could not have come from the bile) its presence in the body always indicates the previous effusion and decomposition of blood.

White Corpuscles of the Blood.—These are larger than the red; they are, when seen out of the body, and dead, spherical masses, with a somewhat granulated surface. In their interior may be seen granules, or sometimes indistinct nuclear bodies. On the addition of acetic acid sometimes a single nucleus is shown, sometimes two, three, or four; sometimes an oval or double nucleus, which appears to be in process of division. It is important to remember that these differences are normal, and not in themselves indicative of disease. When observed at the temperature of the body these cells show lively movements, throwing out and withdrawing portions of their substance, and undergoing various alterations of shape. In the characters which have been mentioned, as in others, they agree with lymph corpuscles, and with the corresponding elements of mucus and pus.

The chemical constitution of the white corpuscles is not of any importance for our present purpose. We will only call to mind the contrast which is observed between the mineral constituents of the corpuscles generally and those of the *liquor sanguinis*. According to Schmidt the relative quantities of metals and acids are as follows:—

	K.	Na.	PO_5.	Cl.
Corpuscles ..	40·89 ..	9·71 ..	17·64 ..	21·00
Liquor sanguinis	5·19 ..	37·74 ..	6·08 ..	40·68

It will be seen from this that the greater part of the potassium and the phosphoric acid are contained in the corpuscles; most of the sodium and chlorine in the liquor sanguinis. This difference is sometimes not without pathological importance.

PATHOLOGICAL CONDITIONS OF THE BLOOD CORPUSCLES.

Variations in Number.—With respect to the red globules, it is certainly proved, that their amount is, *cæteris paribus*, proportionate to the vigour, health, and strength of the individual. In the examinations made by MM. Andral, Gavarret, and Delafond of the blood of various animals, it was constantly observed that those which possessed most strength and vigour, and were generally the finest specimens of the race, gave the highest figures in a series showing the relative amounts of globules; while those that were debilitated and poor showed a corresponding deficiency in this particular. Also, when the breed of a species was improved by crossing it with another, there was a corresponding increase in the quantity of red particles. In the human subject the comparison of the general vigour and activity of the sanguine temperament, both as regards body and mind, with the sluggishness and dulness of the lymphatic temperament, or with the languor and debility of the anæmic patient, shows that the same rule holds good.

Increase of Red Corpuscles.—Plethora is the chief pathological condition in which an increase of red globules has been observed. One of the cases of cerebral congestion mentioned by Andral presented an amount of 138·6 parts per 1,000, an excess of 11 above the normal figure; after venesection the globules in this case were so far diminished that the quantity only amounted to 101·1 per 1,000, considerably below the mean. In various febrile diseases an augmentation of the globules has also been observed; thus in the period immediately preceding the outbreak of continued fever their amount was once found as high as 157·7; in the early period of a case of severe inflammatory fever, the fourth day of the disease, the globules had attained the extraordinary height of 185 parts per 1,000, the greatest amount ever observed; in several cases of typhoid fever (fever with intestinal complication) the globules had risen to 142 or even 149, and even on the second bleeding were found still considerably above the mean; in scarlatina and in measles an increase in the amount of globules was also found, the maximum (which existed in the latter) being as much as 146. No increase was observed in cases of variola or of modified smallpox. A much increased proportion of red globules has been observed in cholera and other diseases in which a rapid drain of liquid from the blood occurs. The *rationale* of this is plain.*

Some writers, especially Vogel, have drawn a distinction between relative and absolute variations in the number of red globules. Relative variations are such as have already been spoken of; absolute variations are alterations in the total number of globules present in the body. The latter are evidently the more important, since the functional activity of the blood must depend on the absolute amount, and not on the proportion of red globules; but absolute determinations are only possible when we know the absolute amount of blood in the body—a matter of which we shall speak hereafter. The condition of the general system co-existing with, and probably occasioned by, the increase in the amount of red globules is exaltation of the animal heat, heightened sensibility, and muscular irritability: the spirits are high, and the mental energy great, the pulse beats full and firm, the power of resistance to debilitating and morbid influences is considerable, the tendency in disease is to active inflammation and high febrile excitement, and bleeding, if employed, is well borne. The effect of iron in promoting an increase of the red globules is well known, but it will often fail where the attendant circumstances are unfavourable; and we have seen a much more rapid effect produced by the change from a scanty to an ample diet. Free exposure to fresh air and light seems also powerfully to promote the formation of red blood, as much as the deprivation of them tends to destroy it.

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* It should be observed that the methods of analysis by which these numbers have been obtained are far from trustworthy. They profess to give only the weight of dried corpuscles, and the exact relation of this to the real weight of the corpuscles is not known. A more exact though exceedingly laborious method, is that of counting, introduced by Welcker. According to this observer the blood of men contains in one cubic millimetre 5,000,000 red globules, that of women, 4,500,000, on the average. If the number exceed 5,500,000 for the male or 5,000,000 for the female; or again, fall below 4,500,000 for the male or 4,000,000 for the female, the condition must be regarded as morbid.

Deficiency of Red Corpuscles.—The opposite condition to plethora for which the term *spanœmia* (*σπανος*, scarce) is more appropriate than anæmia, is essentially characterized by a deficiency in red globules. This condition is met with in the disease known as chlorosis, when there is a chronic deficiency, both of the red and the white globules, and of the solid constituents of the blood generally; so that the proportion of water is much above the normal. Deficiency of red globules perhaps never occurs without deficiency in some other constituents of the blood also. Extreme cases of spanæmia are by no means unfrequent, and, which is not sufficiently known, are by no means unattended with serious danger. Sudden death has in several instances taken place apparently from the cessation of the heart's action, the debilitated organ being insufficiently stimulated by the impoverished blood. In an extreme case of chlorosis Andral found the globules at so low an amount as 38·7 per 1,000, the water at the same time being increased from 790 to 868·7. Similar alterations were found in the blood of other individuals who had become anæmic from other causes, from lead poisoning, the cancerous or tuberculous cachexia, or from repeated losses of blood. In Bright's disease there is evidently a marked failure in the power of producing red globules, and the same is the case in the peculiar affection termed leucocythæmia, which either depends on, or is coincident with, great hypertrophy of the spleen. We are inclined to think that in spanæmic states the red globules are not only deficient in number but defective in quality; they appear under the microscope manifestly paler than those of persons who have a healthy colour, their hæmatoglobin being in all probability not properly formed. The well-known symptoms of this condition are general debility, diminished temperature, palpitation, often excessive, of the heart, and various nervous affections.

Dissolution of Blood Corpuscles.—In some of the most malignant fevers the blood corpuscles appear to be actually destroyed. Dr. Williams has observed this in a case of albuminuria proving fatal by purulent infection, and in a case of malignant scarlatina; we have examined the blood a few times in persons dying of such diseases, but have not found any noticeable alteration in the globules. Unless in very extreme cases, such as that observed by Dr. Williams, it would be very difficult to get clear evidence of the dissolution of blood corpuscles, although it is probable that this may occasionally occur from the operation of some one of the numerous agents before mentioned. It should be remembered, however, that the first effect of all these agents is to separate the red colouring matter from the globules, and dissolve it in the *liquor sanguinis*, and this change has often been observed.

When this is the case the walls of the vessels, and the tissues in immediate contact with them, very commonly become saturated with a red colour, which must be carefully distinguished from that which accompanies inflammation. The effect of diluting the liquor sanguinis upon the blood globules is exceedingly well shown by an experiment performed by Mr. Lane. An animal was bled, and the serum, after the formation of the clot, was as usual colourless, or of a light yellow; a certain quantity of matter was then injected into the veins, and soon after blood was again drawn. The serum of this, however, was of a decided red, contrasting strongly with that of the preceding quantity. It was, therefore, quite clear that the hæmoglobin had been removed from the globules, and dissolved in the serum. We have observed the same thing with the microscope in the blood of frogs, and in that of fœtal vertebrata. When the globules have not been exposed to the action of water, or only in a slight degree, the whole mass is permeated with red fluid; but when water has been freely added, the red fluid entirely disappears. It seems very probable, as Dr. Williams suggests, that the instances of sudden death occurring immediately after copious draughts of cold water in an exhausted state of the system, have been in some measure owing to such alterations of the blood globules as we have just noticed. The same changes may also be concerned in the phenomena consequent on excessive losses of blood, especially in occasioning the local congestions which are apt to take place in them, and in the anæmic generally. Rokitansky mentions that he has observed in a septic state of the blood an altered condition of the globules, which are swollen up from their natural disc-like shape, and have parted with much of their colouring-matter to the surrounding fluid.

The same thing was observed by Kühne in the blood of dogs, in whom artificial icterus had been produced by the injection of salts of the bile acids into their veins; but there is no similar observation on record in cases of icterus in the human subject. Separation of the colouring matter also occurs, locally, in almost all cases of stagnation of blood, as from obstruction of vessels; it also accompanies for the most part actual extravasation. In the disease known as purpura the colouring matter alone appears sometimes to transude, though more generally there is extravasation of the blood globules also; and in many purpuric or petechial patches met with on the surface of internal organs after death colouring matter alone, without any organic elements, is met with. When this condition of the blood is observed after death it is important to bear in mind that it may be a sign merely of very rapid decomposition of the blood, and have the same significance as the livid patches seen on the skin along the course of veins or elsewhere. This too rapid decomposition may be regarded as a morbid phenomenon, but it does not necessarily show that the same condition existed during life. The disease in which this "dissolved condition of the blood" has been most constantly observed is diphtheria, but it occurs also in some cases of pyæmia, of typhus, and yellow fevers, and doubtless of other febrile complaints.

The red globules of human blood exhibit a tendency to cohere together in such a manner as to form tolerably regular piles, or rouleaux; in the healthy condition the cohesion soon ceases, and is not nearly so strongly manifested as in the inflammatory state. Of this, indeed, it is quite characteristic that the globules form rows of some length, made up of numerous discs cohering together by their surfaces, and having their edges disposed so as to form a tolerably straight line. The cause of this tendency is not certainly ascertained, but Dr. Norris, of Birmingham, has shown that the cause must be physical rather than physiological, since discs of various materials, floating in a liquid, exhibit the same tendency. It is, however, essential that the discs should previously be wetted with a liquid antagonistic to that in which they are submerged.

Causes of Variation.—While so much uncertainty prevails respecting the origin of the red corpuscles, it is not possible to point to any foregoing condition as specially tending to promote their growth and increase, or to occasion their atrophy. All that can be said is, that a proper constitution of the liquor sanguinis is certainly essential, as being the material out of which these floating cells are nourished and built up. If this be impoverished, or otherwise deteriorated, the corpuscles will not be properly developed; and again, by improving the quality of their plasma, their healthy condition will be restored. Of this we have a good example in those cases of chlorosis where the administration of iron is sufficient to reproduce the ruddy hue of the complexion. In other cases, a defective state of nervous influence, proceeding from some mental affection, occasions the atrophy of the red particles; but we cannot tell whether this cause acts upon them primarily, or, as is more probable, through the medium of other organs and functions. There can be no doubt that the blood globules have an appointed period of existence, after which they naturally decay. This decay probably takes place in the general course of the circulation, at least in part. Evidence, however, has recently been adduced to show that the spleen is especially the seat of a destructive process, affecting the globules; and that the yellow pigment matter, so frequently found in this organ, is, in fact, the remains of their altered blood-pigment. In the liver also the blood globules seem to yield up their colouring matter, to furnish the yellow pigment of the bile; and the colouring matter of the urine is no doubt derived from the same source. The circumstance that so much pigmentary matter should by these two channels be ejected out of the system shows, on the supposition that it is derived from that of the red globules, how rapidly the latter must undergo decay; and, by consequence, how fast their reproduction must take place. It is worth remarking that one drug, the most commonly used, perhaps, of all—viz., mercury, seems to have almost as much tendency to cause the destruction of red corpuscles as iron has to promote their formation.

PATHOLOGICAL CONDITIONS OF THE WHITE CORPUSCLES.

An increase in the number of the white corpuscles has been observed in several morbid as well as physiological conditions. It will be instructive to consider first those variations which occur in a state of health. It is found that they are present in larger proportion after food has been taken, and are much diminished in number (as compared with the red corpuscles) by prolonged fasting. The maximum seems to be attained about half an hour after meals. A decided increase has been observed in pregnancy, and also during menstruation. After considerable loss of blood the proportion of white corpuscles is also greatly increased; and the same thing has been observed in several diseases both acute and chronic. Among the former must be mentioned inflammations in general, and especially those of the lungs; typhus and typhoid fever; ague, and many so-called pyæmic conditions, as well as more especially puerperal fever. Several of these cases have been described as cases of the occurrence of pus in the blood. According to other authorities, in many long wasting diseases a disproportionate number of white corpuscles may be seen; but in such cases (as in some which have fallen under the observation of the editor) it is quite impossible to distinguish between an increase in the white corpuscles and that diminution in the red of which the general pallor and anæmia give evidence. This is, however, by no means true of the remarkable disease called leuchæmia or leucocythæmia, to which we shall recur presently. The moderate increase now spoken of has been termed *leucocytosis.*

There does not appear to be any positive instance of diminution of the colourless corpuscles.

PATHOLOGICAL CHANGES OF THE LIQUOR SANGUINIS.

This fluid consists of a watery solution of certain inorganic salts, in which there are besides dissolved a large proportion of organic substances. These are distinguished into fibrine, albumen, extractive matter, and oil. We shall consider each of these separately.

When fresh-drawn blood is left to itself it very soon, as we know, passes from a liquid state into that of a solid mass—it coagulates. This change can be conclusively shown to be occasioned not by the corpuscular elements cohering together, as was once supposed, but by the solidification of one of the constituents of the fluid portion of the blood. Müller's experiment of filtering frog's blood, so as to separate the corpuscles, was decisive as to this point. The substance which thus spontaneously solidifies is termed fibrine, and the name evidently has reference to an inherent tendency which it possesses of assuming a fibrous arrangement. Fibrine, when separated from healthy blood, is a tough, tolerably firm, elastic, stringy, whitish-grey looking substance. It is insoluble in water, and sinks in this fluid, or even in the serum from which it has been separated. Under the microscope it appears as an homogeneo-granular or basis substance, with more or less marked tendency to fibrillate, or form actual fibres. The white corpuscles of the blood are very commonly seen imbedded in the fibrinous mass, but they do not appear to contribute to modify its character. This, at least, is true of healthy fibrine; of diseased we shall presently speak. Lehmann describes as follows the actual process of coagulation of the fibrine as seen under the microscope. He says: "There appear here and there individual points or molecular granules, from out of which very soon extremely fine straight threads spring, which go off in a radiating manner from that point, but do not form star-shaped masses, as in crystallization; these threads elongate themselves gradually more and more, and cross with those which have proceeded from other solid points, so that at last the whole field of view appears as it were covered over with a fine, but somewhat felted network. Afterwards this network still proceeds to thicken, and the colourless corpuscles imbedded in it are often scarcely perceptible." With regard to its chemical composition Lehmann remarks very properly that fibrine, such as we obtain, cannot by any means be considered as a pure substance; it must contain the white corpuscles, and probably some amount of albumen and extractive matter, as well as salts. The nearest approximation to an exact analysis gives—

C	52·6
H	7·0
N	17·4
O	21·8
S	1·2
	100

while the albumen of the blood serum contains—

C	53·5
H	7·0
N	15·5
O	22·4
S	1·6
	100

So that it must be regarded as belonging to the general group of albuminous substances. Fibrine is hardly known in solution; and should probably not be regarded as ready formed in the blood, but rather as formed at the moment of coagulation, by the combination of two substances called respectively the fibrino-plastic substance or paraglobulin, and the fibrinogenous substance or fibrinogen (Schmidt). The former is like, though not quite identical with, globuline, or the albuminous substance contained in large quantity in the red blood corpuscles. The latter is also an albuminous substance, and is contained in all serous fluids, such as those of the pericardium and pleura, hydrocele fluid, &c., and also in blood serum. These substances artificially prepared and artificially mixed produce a coagulum absolutely identical with natural fibrine. Spontaneous coagulation accordingly must depend on the combination of these two substances, though it is not clear why they do not always combine. For a discussion of these questions we must refer to books on physiology.

USES OF FIBRINE.

One very important end which the fibrine serves is the formation of coagula at the orifice of wounded vessels, preventing the occurrence of further hæmorrhage; and another is to form a temporary uniting medium of the sides of wounds. But a still higher dignity has been assigned to the fibrine; it has been regarded very generally as the plastic material, *par excellence*, from which all the tissues are formed, and the small amount of it present in blood was supposed to show that it was constantly being drawn off for the nutrition of the tissues as fast as it was formed. Of late years, however, much evidence has been adduced, which at least goes some way to establish an opposite view, and throws considerable doubt on the correctness of the old opinion. It should never be forgotten that by fibrine is commonly meant, not a chemical substance, but a mass imbedding the red and white cells, the latter of which certainly have some capacity for growth and organization. We will review the arguments on both sides.

In support of the specially plastic and organizable character of fibrine, it is argued that it seems to be by its means that the reparation of wounds is effected. A thin layer of coagulating, fibrillating material is the medium which unites and holds together the divided surfaces, and forms the first organic connection. So also when a fluid containing much fibrine is effused on the surface of serous membranes, it very commonly forms bands of adhesion passing between the opposed layers. These new-formed structures resemble very much normal, white, fibrous tissue. There is evidence to show that layers, and even masses of fibrine, containing leucocytes, are capable of being organized; so far, at least, as to become fibroid tissues, and that vascular networks are developed in them. Andral mentions a case of apoplectic effusion in the subtance of the brain, in which—death having occurred many years after—a mass, of pale red colour and fibrous appearance, and traversed by numerous small blood vessels, anastomosing with those of the brain, was discovered. M. Louis has recorded the occurrence of a vascularized coagulum in a tuberculous cavity in the lungs; and many similar instances have been noticed, in which the fibrine of effused blood has evinced its capacity for organization. But how lowly is this organization, never in any known instance amounting to more than the formation of a fibrous tissue, more or less closely resembling the natural. This, almost of itself, is a proof that fibrine is not the peculiarly organizable and plastic element that it has been considered to be. It may also be said that albumen, which is the only other organizable constituent of the blood, shows no tendency, even when collected in large quantity, as in the fluid of ascites, to pass into any organized form; while fibrine, when effused, does at least assume the appearance of a lowly organized tissue. The condition, moreover, of the fibrine seems to be in some manner an indication of the vigour and health of the system. If it contracts well, and forms a firm, dense clot, there is reason so far to conclude that the constitution is sthenic and unbroken; but if, on the contrary, the clot be soft and easily broken up, the system is probably in an opposite condition. The above statements seem to amount to this, that fibrine is certainly capable of assuming a low type of organization; but they entirely fail to show that it is the special blood constituent which is applied to the nutrition of the different tissues, and that therefore its abundance is a sign of vigour and health.

On the other side, the counter-evidence which we have to adduce is certainly of great force. Bleeding, which we saw to have a powerful effect in diminishing the quantity of red corpuscles, has none such upon the fibrine; nay, it rather seemed to tend to increase it. In Andral's ninth case of articular rheumatism the fibrine at the second bleeding was 7, and at the third 6, while in the first it was 5·4. In the tenth case at the fourth bleeding it presented the extraordinary figure of 10·2 per 1,000, while in the first it did not amount to more than 6·1. The globules, however, were reduced by the three subsequent bleedings from 123·1 to 101. Starving also, instead of lessening, was found to increase the quantity of fibrine. An increase in this element was found in meagre, half-starved horses, amounting to as much as 7 or 8 beyond the healthy mean; and in one case, where no food was given for four days, the quantity of fibrine was found increased from 5 to 9. On the other hand, the improvement of a species which we found to be marked by an increase in the proportion of globules, seems to be also characterized, though less strongly, by a diminution in the quantity of fibrine. The average quantity of fibrine in a flock of sheep of pure blood was determined by Andral to be 3·1; in a cross-bred flock, the average was only 2·8. The blood of the fœtus and of the new-born animal, in whom certainly development and growth are proceeding rapidly, and in whom there must, therefore, be a constant demand for plastic material, is rich in globules, but poor in fibrine. Andral found in lambs, during the first twenty-four hours after birth, a proportion of fibrine only amounting to 1·9 per 1,000; at the end of the fourth day it had risen to 3 parts. This observation, as well as the familiar effect of diseases preventing the due oxygenation of the blood, in causing it to remain fluid after death, which seems to imply the non-formation or destruction of the fibrine, points very clearly, as it seems to us, to the conclusion that fibrine is an oxidation-product, and rather belonging to the descending series of destructive assimilation, than to the ascensive, plastic, and formative.

That fibrine takes an important part in the reparative process cannot be doubted; we constantly find it forming the uniting medium between divided parts, but have we any evidence that it becomes further developed, and passes into the form of any tissue more highly organized than that of the cicatrix? Surely there is not the least, or rather all that we know of the process of reparation tends to contradict such an idea. The case of a divided nerve serves to illustrate this point very well. Within a short time after the operation the cut ends of the nerve are united together by fibrinous effusion, which has solidified round them; this passes afterwards on into the form of imperfect fibrous tissue, and so the apparent continuity of the nerve is restored. But we know that it is not really restored until very much later; we know that many months must elapse ere the severed nerve-tubules can be again connected by their own proper tissue, and their function restored. Now, in this really reparative act there is no reason to believe that fibrine takes any prominent part, that it is at all more concerned than the albumen and oily matter of the blood, which are the elements, chemically considered, of which the nerve-matter actually consists. In the same way, in every organizing act in which there is more than the mere coagulation of fibrine, albuminous serum is also present, and we have no reason to exclude it from participating in the formative process. Once more, if we take instances where deposition of fibrine has almost exclusively taken place, do we find development and growth proceeding actively in these deposits or not? The wall of an aneurismal sac is often lined with dense layers of coagulated fibrine, and yet Professor Simon testifies that on the most careful examination of these layers no trace whatever is to be found of new organization. So it is with the masses of fibrine that are deposited in the spleen, the kidney, and in other parts. They show, after a time, a tendency to retrograde and dissolve, but none to develop into any higher grade. The small, fibrinous, sub-pleural nodules often seen in tolerably healthy lungs, are favourably circumstanced as regards vascular supply, for growth and further development; but they do not appear to enlarge or manifest any vital activity. The deposits of fibrine upon the valves of the heart, the wrongly-called vegetations, in like manner, show no innate capacity of development and growth; they tend to contract and harden, or to become penetrated with calcareous matter, and never show any organized arrangement beyond a low grade of fibrousness.

The circumstance which seems to have contributed most, or at least very greatly, to establish the opinion of the highly plastic quality of fibrine is, that its quantity is found to be so very largely increased in sthenic inflammatory diseases. Not only its appearance in the thick, buffy coat, but the quantity of solid effused matter, forming layers of false membrane, of considerable firmness and thickness, together with the character of the attendant symptoms, inspired an idea that the substance thus abounding in these diseases of marked sthenic type, and so called increased action, and comparatively deficient in affections of asthenic type, was that which was especially plastic, and employed in the construction of all the various tissues in healthy nutrition.

While thus rejecting the doctrine that fibrine is the sole or chief plastic element, we do not wish, on the other hand, to deny its importance, when of proper quality, in maintaining the due consistence of the blood, and in fulfilling such other purposes as those at which we have glanced. We now proceed to consider the variations of this substance, in different morbid conditions, as to quantity and quality.

VARIATIONS IN FIBRINE.

The average for the fibrine of ordinary venous blood, adopted by Andral, is 3 parts per 1,000, but most observers place it rather lower. Scherer found in the blood of healthy men 2·03—2·63 parts. Its quantity, as mentioned, is greater in arterial than in venous blood, and is said by Schmidt, after numerous examinations, to be only one-third in portal venous blood of its ordinary amount in that of the jugular vein. Sex does not appear to affect the proportion of fibrine, but the state of pregnancy does materially; in the first six months the quantity is decreased, the average being 2·3; during the last three months the quantity is increased, so as to average 4. After parturition the quantity for a time seems to be still further increased, a circumstance which may have some relation to the tendency to uterine inflammation and mammary abscess, which marks this period. In very early infancy the quantity of fibrine in the blood appears to be small, but it experiences a marked increase at the period of puberty. Lehmann, in experiments upon himself and Nasse, in experiments upon dogs, found that animal diet increased the proportion of fibrine above that which was found under a vegetable diet.

Increase of Fibrine.—Passing now to the morbid conditions in which the fibrine is found increased, we find, as a general law, that in all inflammatory diseases especially this is the case. In acute articular rheumatism, Andral records in one case as high a proportion of fibrine as 10·2 per 1,000, in several others it amounted to 6, 7, 8, or 9. In bronchitis (acute) the highest figure obtained was 9·3, in pneumonia 10·5. The maximum in pleurisy was 5·8, in peritonitis 7·2. The increase was nearly the same in one case of erysipelas, in which it amounted to 7·3, and in another of tonsillitis, in which it was 7·2. A very important circumstance, well shown by Andral's tables, is, that the effect of bleeding was not to diminish the fibrine; in this respect there was a marked contrast between the fibrine and the red globules; the latter sunk with each successive abstraction of blood, while the former most often rose, sometimes considerably. The following examples of this fact are very striking:—

	1st bleeding.	*2nd.*	*3rd.*	*4th.*	*5th.*	*6th.*
Acute Rheumatism	6·1	7·2	7·8	10·2	9	7
Pneumonia . . .	7·1	8·2	9·0	10·0		
Peritonitis . . .	3·8	4·7	6·1			
Pleurisy	3·9	5·8				

In tuberculization of the lungs the fibrine shows a decided increase, which, however, is most marked when intercurrent inflammation is set up in the part. In the crude state of the tubercles the mean of the fibrine is about 4; when softening has commenced it is about 4·5; when cavities have formed it is from 5 to 5·5. The red particles steadily decrease from the first. In chlorotic persons the quantity of fibrine is maintained at its usual average, and is sometimes a little above; this circumstance, taken together with the great diminution of the globules, accounts for the formation of a buffy layer on the surface, which is not uncommon in such blood.

Deficiency of Fibrine.—This is observed in very various morbid conditions. If we take as a certain indication the non-coagulated state of the blood (which, perhaps, we are warranted in doing), it seems to be very deficient, generally, in all diseases proving fatal by asphyxia, or in which the respiration has been considerably interfered with for some time before death. Thus in cases of obstructive heart disease, the blood is very commonly in a fluid state, or very imperfectly coagulated, the coagula, such as they are, being very soft, and extremely friable. The same state is observed in cases of cyanosis, in which, owing to mixture of the venous and arterial blood, this fluid is never properly oxygenated. Excessive fatigue is said to prevent the blood from coagulating, but this has been contradicted by Mr. Gulliver, who found the blood coagulated in a hunted stag, and in two hares run down by harriers. Various poisons seem to have the effect of preventing coagulation of the blood; among these are hydrocyanic acid, carbonic acid, sulphuretted and carburetted hydrogen. Andral states that if a concentrated solution of carbonate of soda be injected into a vein the animal presents the symptoms of typhus or scurvy (*i.e.*

of a blood disease), and the blood is found fluid in the vessels. This statement is confirmed by Mr. Blake, and the same result proved to be produced by many other substances; others again, though of very analogous character, produced a contrary effect. For instance, when caustic soda or carbonate of soda was employed, the blood was coagulated imperfectly, or not at all; but when liquor potassæ or its carbonate was used, the blood coagulated firmly. We have ourselves observed the formation of a buffy layer on the blood of an animal who had taken liquor potassæ for several days, to the extent of disordering its health. Nitrate of potash and many other neutral salts did not at all impede the coagulation of the blood, while arsenious and oxalic acid, infusion of digitalis, and some metallic astringent salts did so decidedly. We may infer from these observations that it is not the alkalies, as such, nor the neutral salts, as such, which produce the effects that are usually ascribed to them, upon the blood, but certain substances of particular qualities. Again, in adynamic fevers, we often find the blood remarkably fluid, so as to gravitate after death to all the depending parts, and during life probably occasioning a tendency to hæmorrhages, petechiæ, and vibices, which occasionally take place. The deficiency of fibrine in these cases is confirmed by analysis. Andral noticed some diminution of this element in the outset of continued fevers. It never increased except on the supervention of an inflammation, and in the height of the disease sometimes sunk very low. In two cases, on the fifteenth day, it did not amount to more than 1 per 1000. A deficiency of fibrine might have been anticipated to exist in purpura hæmorrhagica; and, indeed, sometimes this seems to be the case. Simon, in his "Animal Chemistry," gives two analyses, in one of which it is mentioned that there was no fibrine, in the other it only amounted to 0·905. Dr. Watson, speaking of this, or of the allied disease—scurvy—quotes a case, recorded by Huxham, in which "neither of the portions of blood that had been drawn separated into serum and crassamentum as usual, though it had stood many hours, but continued, as it were, half-coagulated, and of a bluish-livid colour on the top. It was most easily divided on the slightest touch, and seemed a purulent sanies rather than blood, with a kind of sooty powder at bottom." Dr. Copland and others refer to similar cases. On the other hand, Dr. Budd testifies that in some cases of scurvy, the coagulation takes place as in healthy blood; and in two cases lately examined by Dr. Parkes, it appears that the fibrine, at least, was not in any great degree diminished.* Dr. Graves also mentions a case in which, after each of three bleedings, there was formed a firm coagulum, with a buffy coat. The plethoric condition, characterized as we have seen by an excess in the quantity of red globules, and evidencing a tendency to congestion and hæmorrhage, is considered by Andral as generally associated with a deficient proportion of fibrine. He found in a strong, athletic man, who had symptoms of cerebral congestion, as low a figure of fibrine as 1·6, and remarks that the minimum quantity occurred in those cases in which the symptoms of congestion were most intense. In a female, who had been struck down senseless by an attack of apoplexy, the first bleeding showed the small proportion of 1·9 of fibrine, the globules being at 175·5. After three days, when consciousness had begun to return, she was bled again; and now the fibrine amounted to 3·5, while the globules had diminished to 137·7.

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* In five analyses performed by Becquerel and Rodier the general results were as follow:—The clot was always of good consistence, the density of the serum low, the water increased, the quantity of globules diminished as well as their ferruginous contents; the fibrine was never diminished—sometimes increased: there was no increase of alkalinity or of salts; the solids of the serum were notably diminished.

Quality of Fibrine.—Alterations in the quality of the fibrine manifest themselves very clearly in the varying size and firmness of the coagulum, which forms in blood drawn from a vein, as well as in the peculiarities of structure which microscopic examination reveals. We will consider the general and the textural differences separately. To estimate aright the condition of the coagulum of the blood a full stream should be allowed to flow from a sufficient orifice into a deep vessel, which should be afterwards covered over, and should have been previously warmed. If drawn in a small, trickling stream, and received into a cold, shallow vessel, or if subsequently agitated, the coagulation is disturbed, and takes place either too rapidly, or forms, in the case of being agitated, irregular shreds.

The coagulum, when formed, may be very large and firm, so as to offer considerable resistance when an attempt is made to divide it. This implies a fair proportion, or, perhaps, rather increased, of healthy fibrine, with a considerable amount of red corpuscles. I have been informed of the case of a plethoric female whom it was necessary to bleed frequently during her pregnancy. The coagulum was described to me as so firm "that it might have been kicked from one end of a room to the other." On the other hand, the coagulum may be large, but so lax as to be very easily divided, and, if handled, readily breaking up. This implies either a deficient quantity of fibrine, or a defective contractile quality, or most commonly both. It may generally be taken as a positive sign that bleeding is not necessary, and that it will not be borne well. The coagulum, again, may be very firm, but considerably shrunken and contracted, manifesting this not only by its recession from the sides of the vessel, but by the concavity of its upper surface, which at the same time is covered with a layer, more or less thick, of a light-yellow colour. This layer is fibrine separated from the red corpuscles, and is commonly termed the "buffy coat." In this case the quantity of fibrine is much increased, and probably also its contractile quality, which occasions the reduced size of the clot, and the drawing in of the surface, or, as it is called, "the cupping." A buffy coat sometimes forms on the surface of clots which are rather deficient in firmness; it is, however, but thin, rather transparent, and produces no "cupping." It is mostly seen in rather asthenic conditions of the system, and from its appearance the blood which presents it is distinguished as "sizy." A very small, firm clot, with a more or less buffy surface, indicates a diminution of the red corpuscles, and at least a relative excess of fibrine.

The following circumstances * are favourable to the formation of the buffy coat:—(1.) Slowness of coagulation, which gives the red corpuscles more time to sink. (2.) Increased weight of the corpuscles, and their aggregation together into rouleaux. (3.) A diminution in the specific gravity of the serum. (4.) A great diminution in the proportionate quantity of the red corpuscles, or an increase in that of fibrine. None of these circumstances, however, seem to us to account fully for the phenomenon in question; and we are much inclined to believe, with Dr. Alison, that there exists an absolute tendency to separation between the fibrine and the corpuscles, somewhat, perhaps, of the nature of that which prevents the commixture of some dissimilar fluids. He remarks: "1st. That the formation of the buffy coat, though no doubt favoured or rendered more complete by slow coagulation, is often observed in cases where the coagulation is more rapid than usual; and the colouring matter is usually observed to retire from the surface of the fluid in such cases before any coagulation has commenced. 2nd. The separation of the fibrine from the colouring matter in such cases takes place in films of blood, so thin as not to admit of a stratum of the one being laid above the other. They separate from each other laterally, and the films acquire a speckled or mottled appearance, equally characteristic of the state of the blood with the buffy coat itself." It does not seem necessary to assume that there is any actual repulsion of the red particles from the fibrine, the tendency to separation may simply depend on the increased attraction (which is manifest) of the respective parts of each element together.

The differences in *textural quality*, which fibrine often presents, have been admirably described by Rokitansky and Mr. Paget. As, however, the former seems to have altered his opinion, we omit the description he has given, though our own observation inclines us to think it is substantially correct.

Professor Paget, agreeing closely with Rokitansky's previous account, expresses the same facts more shortly and simply. His description refers, however, to fibrine, as it appears in exudations; while that of the German pathologist is expressly confined to the fibrinous coagula which are found within the vascular system; and the variations may therefore depend upon the greater or less facility with which the white corpuscles leave the blood vessels. The correspondence, however, between the fibrine *in* and *out* of the vessels is so close that the characters of the one apply also to the other; and we may therefore say that all the varieties of the fibrine of the blood, as manifested by the condition of the coagula, depend upon the predominance of one of two forms of structure. In healthy fibrine the fibrous structure greatly predominates, the whole mass fibrillates more or less perfectly, and the included corpuscles are comparatively few. This is Mr. Paget's *fibrinous* variety. Unhealthy fibrine, which tends to disintegration, consists of a granular mass, imbedding very numerous nuclei and cell-formations; this is the *corpuscular* variety. These two correspond to Rokitansky's first and fourth varieties; and his second and third are only combinations of them in different proportions.

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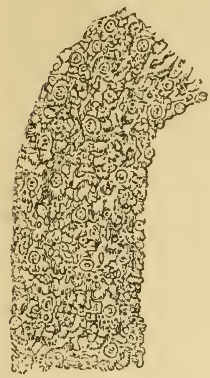
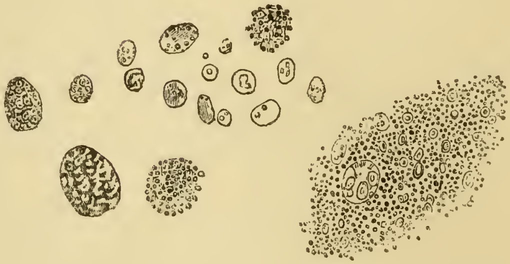
* Dr. Todd and Mr. Bowman's "Physiological Anatomy," vol. ii. p. 295. It must be remarked, however, with regard to diminished density of the serum, that Mr. Gulliver has shown that in serum rendered thicker, heavier, and more viscid, by the addition of mucilage, the red corpuscles subsided not only more rapidly, but also more completely, than in serum, which was rendered thinner, lighter, and less viscid, by being mixed with a saline solution. Mr. Wharton Jones—from whom we have, in part, taken the above passage—believes that the viscid state of the liquor sanguinis promotes the subsidence of the red corpuscles, by increasing their natural tendency to aggregate together.

Fig. 1.



Corpuscular unhealthy fibrine, from exudation on pericardium. It consists of an homogeneous granular basis, imbedding numerous corpuscles.

Fig. 2.



Softening fibrine from a vein clot. The dark points are minute oil drops.

CHANGES OF BLOOD CLOTS.

We shall here briefly notice the metamorphoses which blood-coagula may undergo:—(1.) A more or less perfect development of fibre may take place, in which the corpuscles are chiefly concerned. (2.) The coagula may fall to pieces, and undergo a kind of dissolution into a pappy or pus-like fluid, or in some cases into a fluid which is really of the nature of pus. This change may befall coagula of healthy fibrine, in consequence of their being placed in conditions unfavourable to fibre-development, but is more particularly observed in the corpuscular varieties. It was shown by Mr. Gulliver that the puriform fluid often found in vessels was really fibrine which had undergone softening; and it was an important step to prove that such collections of pus-like matter were not the result of phlebitis; but in recognizing their non-inflammatory origin, perhaps, it has not been seen clearly how very similar, or even identical, they might be with certain forms of true pus. (3.) The fibrine may part with some of its natural moisture, and change into a resisting, stiff, dull, translucent, or also opaque, horny mass. In process of time it may become calcified. (4.) It may undergo fatty transformation, becoming converted into a mass of small, oily molecules or drops. (5.) It may gradually be dissolved and taken up again into the circulation. Rokitansky mentions, as an instance of this, the removal of fibrinous vegetations from the cardiac valves.

CHEMICAL CONSTITUENTS OF THE SERUM.

Albumen is the principal nitrogenous constituent of the serum, in which it exists, dissolved in water, in the proportion of 63 to 72 parts per 1,000 of blood. The specific gravity of the serum is on an average 1·028, and varies less than that of the entire blood. The serum is naturally of a light yellow colour, which does not appear to depend on the presence of hæmoglobin or of bile pigment, but to be special to this fluid. We have already expressed our dissent from the doctrine that the fibrine is the sole or chief material intended for the growth and nutrition of the tissues, and fully believe that the albumen is quite as much or more applied to this purpose. We have no means at present of ascertaining numerous qualitative variations which probably affect the albumen of the serum. Indeed we cannot but believe that its composition must be liable to continual minute changes, as on the one hand nutritive material for various tissues is drawn off from it, and on the other, chyle, scarce yet raised to the blood-standard, and lymph, containing effete or semi-effete residua of nutrition, are poured into it. All such, however, to a certain extent, are clearly natural; and were our powers of analysis greatly more refined, we should probably find that it was by the most gradual steps that physiological variations passed into morbid.

Increase of Albumen.—The amount of albumen, according to Andral and Gavarret, is notably increased in various diseases; but this excess does not appear to be characteristic of any. In acute rheumatism an increase was found varying from 4 to 24; in pneumonia, the highest increase was about 12; in pleurisy, the extraordinary amount of 34 in excess was once observed; and several other instances are mentioned of lower degree. Peritonitis, tonsillitis, and erysipelas all furnish cases in which there was more or less considerable increase of the albumen. This is the case also in tubercular disease of the lungs, and in simple and continued fevers. Bleeding does not appear to influence the quantity of albumen in a very constant way; on the whole it tends to decrease it; and this seems to be especially the case in typhoid fever. In cases of cerebral congestion and of apoplexy, generally considered as examples of plethora, there was found, especially in the former, a very considerable increase in many cases of the albumen. This amounted once to 24·8 beyond the mean. In the latter there were several instances of marked diminution. Cases of chlorosis, in which the globules are so remarkably diminished, show rarely any diminution of albumen; and sometimes a considerable excess, amounting to 14 or 20 parts per 1000.

Diminution of Albumen.—Diminution of the albumen of the serum probably takes place in various diseases, attended with defective nutrition and wasting, but has been more particularly observed in renal dropsy. It appears as if the albumen of the serum, being drained off in the urine, and in the dropsical efflux, there remains behind a less quantity in the blood. This idea is confirmed by the results of three successive analyses of Andral's, in the first of which there being much albumen in the urine, the serum contained only 57·9; in the second, the urine containing less albumen, the amount in the serum had increased to 66; in the third, the urine being no longer albuminous, the quantity in the serum had returned to its normal figure, 72. In one of our own cases, anasarcous fluid had a specific gravity of only 10·14; it contained much albumen; was weakly alkaline. The patient had morbus Brightii. It is also in accordance with the circumstance noticed by Dr. Bright and others, of low specific gravity of the serum in this disease. Still this explanation does not accord with the general fact, that dropsical effusions are more watery, and contain less albumen than the serum, which one would therefore expect to find of greater density in such cases. Probably the supply of such albumen, both from the chyle and lymph, is defective in quality and quantity. Becquerel and Rodier state that in diseases of the heart the albumen of the serum varies but little, as long as there is no dropsy; then it diminishes, and often considerably. In the disease called the *rot*, affecting sheep, which is characterized by the presence of numerous distomata (flukes) in the biliary ducts, M. Andral and his coadjutors found the albumen of the blood considerably diminished, as well as the red globules, while the water was greatly increased. Sheep are also subject to ordinary anæmia, *i.e.*, to a deficiency of red globules only in the blood, the albumen remaining at its normal amount; and it is very worthy of remark, that in these latter cases dropsy does not take place, while in sheep affected with the rot it is not uncommon. This seems to point out that when in cases of cachexia and debility serous infiltrations of the limbs occur, it is owing to a diminution in the quantity of albumen in the blood.

EXTRACTIVE MATTERS.

Chemistry has as yet ascertained too little respecting these substances, even in the healthy condition, to make any conclusion possible regarding their variations in disease. Simon's division into water extract, proof spirit extract, and alcohol extract is of no avail for physiology or pathology. As his eminent namesake remarks, what we want is a division of these matters according to the organs or systems of organs that produce them. Still the recognition of the existence of such matters in the blood is important, as showing us the actual presence of principles that are effete, or tending to become so in this fluid, and reminding us how often ill-health and *malaise* may depend on the formation of unnatural products of this kind, which come at length to be generated by an almost habitual vice of the system.

According to Lehmann's estimate, the quantity of extractive matter in healthy blood is 0·25 to 0·42 per 100. Nasse found a larger proportion in the blood of children and of young animals than in that of adults. Arterial blood, according to Lehmann, contains more than venous blood in the proportion of 5·374 parts to 3·617. Portal vein blood of horses, five to ten hours after food, contained on an average 7·422 parts; twenty-four hours after food the quantity amounted to 10; but it was always less than that existing in hepatic vein blood, which averaged above 18. The existence of a larger quantity of extractive matter in arterial than in venous blood, may perhaps be accounted for by the increased oxydation of some of the organic matters which takes place in and after the passage of the blood through the lungs. The large amount in the blood of young growing creatures is in correspondence with the greater activity of their circulation and their nutritive processes generally. The excess in the hepatic vein above the portal blood indicates that an absorption of matter from the hepatic cells into the current traversing the lobules takes place. Liebig has particularly described three substances which appear to belong to the class of extractive matter, creatin, creatinin, and inosinic acid; the two former seem to be of the nature of alkaloids, the latter combines with bases as an acid. They are all nitrogenized substances, and are found in the watery extract of muscle; the two former are present in the urine, and have been detected in the blood, though in very minute quantity. Creatin, Lehmann states, is analogous to thein, an alkaloid, which in some trials has produced very severe nervous symptoms, even when taken in small doses. May slight chemical changes in creatin or the allied substances render them capable of producing any similar phenomena?

OILY MATTER IN THE BLOOD.

The quantity of oil existing in the blood cannot be estimated only from the amount contained in the serum, for it is present also in the red corpuscles and in the fibrine. The quantity contained in the crassamentum, which is made up of these two components, is not much inferior to that in the serum. The serum of arterial blood contains less oily matter than that of venous. Lehmann gives 0·264 per cent. as the proportion in the first, 0·393 per cent. in the latter.* Chevreul gives the quantity of oily matter in blood-clot as amounting to 4 or 4½ per cent. Lecanu distinguishes a crystallizable and non-crystallizable oily matter in the blood, the former in the proportion of 1·20 to 2·10, the latter in that of 1 to 1·30 per 1000 parts of serum. Cholesterine is the only crystalline fatty substance which has been certainly found in blood; it is of very common occurrence in exudations in various parts, and in some tumours; it forms the well-known rhomboid tablets by which, when in a solid form, it is immediately recognized. The blood of females, according to Becquerel, contains on an average more fatty matter than that of men. In both sexes the quantity of cholesterine increases with advancing years, after the age of forty or fifty. It seems established, as was natural to expect, that the quantity of oily matter in the blood increases after taking food. A milky state of the serum had often been observed, but though it was generally supposed to depend on the admixture of chyle, this could hardly be said to have been proved until lately. Dr. Buchanan's experiments upon healthy persons show that the serum "becomes turbid about half an hour after taking food, the discoloration increases during several hours, attains its maximum in about six or eight (after a full meal), and then becomes gradually clearer till its limpidity is restored. The opaque serum is generally milk-white, sometimes cream-yellow, or yellowish brown, like thin oatmeal gruel; or it merely loses its limpidity, and is like weak syrup. It always contains solid white granules, smaller than the blood corpuscles, which are suspended in it, and which will rise in a white cream to the surface, either spontaneously or after the fluid has been saturated with common salt. The cream thus obtained is soluble in caustic potash, but insoluble in ether or alcohol, and is considered by Dr. R. D. Thomson as probably a proteine compound." A permanent milkiness of the serum, independent of variations in the supply of food, must be regarded as a morbid condition. It is especially seen in drunkards. In a case of this kind, which lately came under our observation, this was so strongly marked that a single drop of blood drawn by the prick of a needle became immediately surmounted by a white opaque layer, resembling cream. The microscope showed the serum to be crowded with fatty molecules, presenting an active Brownian movement. They were unaffected by acetic acid; a notable quantity of fat could be extracted by ether. The man's general health was good; he confessed to drinking freely, but was by no means generally inebriate. The microscopic appearance of milky serum, as we have observed it, has depended on the presence of a diffused finely divided matter, much resembling the molecular base of the chyle. Sometimes, however, distinct oil drops are observed. The general result of Becquerel and Rodier's very careful analysis of the blood in various diseases, with regard to the variations in the quantity of oil, is that almost from the outset of every acute disease the amount is increased, and particularly that of the cholesterine. Diseases of the liver, Bright's disease, and tuberculosis have the same effect.

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* Lehmann found in 100 parts of dried blood corpuscles of the ox 2·249 of oily matter; arterial blood corpuscles contained 1·824 parts of oily matter per 100 thereof; venous blood 3·595 parts.

SALINE INGREDIENTS.

There remain for our consideration the various salts of the blood, and the water which holds them in solution. The amount of salts in the blood of man is somewhat greater than in the blood of woman; that of the former contains, on an average, 8·8 per cent., that of the latter 8·1; in both sexes the variations compatible with health are considerable. The blood of adults contains more salts than that of children; arterial blood more than venous. The prolonged use of aliments containing much common salt is said to cause an increase in the proportion of the latter, and of the other salts generally. The following appear to be the principal saline combinations in the blood:—chloride of sodium and potassium, sulphate of potash, carbonate and phosphate of soda. Of the alterations which the salts undergo in different diseases we have not much knowledge. In malignant cholera the excessive drain tells most on the fluid part of the blood, and hence that remaining in the vessels is thick and tarlike; hence, also, the extraordinary, though temporary, effect of injecting saline solutions, which return to the blood the material effused from it, and revive all the functions that were well nigh extinct. Doubtless, if the intestinal discharges could be arrested, the effect would be permanent, but as it is their effect is soon exhausted. Vogel states that "the salts are increased in scurvy, and it is very probable that this change influences the condition of the fibrine, hindering its coagulability, and, perhaps, checking its formation; that it affects the blood corpuscles, by withdrawing their water, rendering them granular, and collecting them in heaps; and that it thus plays an essential part in the disease itself." However, his statement has been contradicted rather than confirmed. In the cases of purpura examined by Dr. Parkes, which have been alluded to, the quantity of salts seems to have been below the average. In acute exanthemata, in dysenteric affections, in endemic agues, Lehmann mentions the proportion of the salts to be increased, as also in Bright's disease, in typhus, and in all kinds of dropsy and hydræmia. In violent inflammation the salts are much diminished. This, however, seems very questionable with regard to sodium chloride.

WATER.

Lecanu's estimate of the mean quantity of water in 1,000 parts of blood is 790; he found more water in the blood of women than in that of men, more in the blood of children and of aged and debilitated persons than in that of vigorous adults, more in the lymphatic than in the sanguineous temperament. It is clear that the whole quantity of water is not contained in the serum, a certain proportion, which must vary with the specific gravity of the blood, is enclosed in the red corpuscles, and holds their colouring matter in solution. The serum of arterial blood and of portal vein blood is said by Lehmann to contain more water than that of venous blood generally. It is a very remarkable circumstance, and strongly indicative of wise provision, that it is very difficult to demonstrate by analysis an actual increase of the quantity of water in the blood after copious drinks have been taken. Denis and Schultz are at issue as to whether such an increase is detectible or not. This seems to show how exactly the vascular system is kept at a certain degree of tension, so that in proportion as absorption at one part takes place, excretion at another ensues correspondingly. The effect of bleeding and starving, which was before noticed, of reducing the amount of globules, tells, of course, proportionally, in increasing the quantity of water; this can easily be ascertained, as it is in great measure relative; the merely positive increase is much more doubtful. Andral mentions a case of confirmed chlorosis, in which the water in the blood amounted to 867·9, an increase of nearly 78 parts per 1000. Lehmann states that in the beginning of most diseases, especially acute ones, the blood is found more watery than natural, the serum, however, at the same time, being richer in solid contents. He accounts for this by supposing that the material which should have been applied to the formation of the globules, or which results from their decay, remains in the serum. During the first ten days of typhus, the first stage of scarlet fever, measles, and cholera, this increase in the watery constituents of the blood does not appear to take place.

In cholera the most striking diminution in the proportion of water takes place; apparently as a consequence of the immense transudation of fluid into the intestines. Other changes are, however, also present; the serum becomes not only richer in albumen and salts, but also appears to withdraw from the corpuscles the salts proper to them, viz., potassium salts and phosphates (Kühne). There are also physiological variations in the percentage of water, which diminishes at first in starvation, but recovers itself if abundant water be ingested. The amount is also diminished by taking much salt.

The condition commonly called anæmia should, it would seem, more properly be named hydræmia, since in most cases it is not so much a deficient quantity of blood, which it is intended to describe, as a defective quality. Perhaps a person reduced greatly by phthisis, or any exhausting disease, is really in a state of anæmia, he has less blood than natural in his body, as well as too watery; but a female suffering from the consequences of amenorrhœa, with pale lips and face, is much more likely to be in a state of hydræmia, the mass of blood not being diminished, but its red corpuscles replaced by water. The excess cannot, however, be sufficient materially to alter the density of the fluid, since the corpuscles preserve their form.

ABNORMAL CONSTITUENTS OF THE BLOOD.

Having examined the variations which the several constituent elements of the blood are liable to undergo, we next proceed to make a few remarks on certain abnormal matters which are occasionally present in it. *Carbonic acid gas*, the product of respiration, *i.e.*, of the conveyance of oxygen throughout every part of the frame, becomes, if it accumulates beyond a certain small amount in the blood, the cause of serious disorder and speedily of death. Various diseases of the thoracic viscera, or impediments to the free action of the walls of the chest, prevent more or less the due oxygenation of the blood, which is indicated by the dusky hue of the complexion, the lividity of the lips, the sensation of oppression at the chest, and of dyspnœa. The larger the quantity of blood which is circulating in the vessels, and the more vigorous the state of health and the general activity of the functions, the greater must be the accumulation of carbonic acid when any asphyxiating cause begins to operate, and the more severely will its effects be manifested. If, however, the mass of blood be greatly diminished by exhausting drains, by diminution of food, and by non-development of its corpuscles, then the amount of respiratory action may be also greatly diminished without producing the symptoms above mentioned. Thus, if a person in health should suddenly be deprived of one-half or three-fourths of his breathing apparatus, he would quickly die, suffocated; but a patient in the last stage of phthisis, whose lungs are destroyed to the same extent, may continue to live on without experiencing any notable dyspnœal distress. In the same way, when during violent exertion, a greatly increased quantity of carbonic acid is formed, the amount of oxygen introduced into the lungs requires to be increased in proportion; and hence one cause of the hurried and panting respiration. A hybernating animal scarcely breathes at all; its animal heat is not above the temperature of the atmosphere, and all its functions are in abeyance; carbonic acid, therefore, is not formed, and the inhalation of oxygen is not necessary;

life, reduced to this low ebb, continues in an atmosphere which would cause instant suffocation if the animal were awake. Man does not hybernate, but it seems highly probable that the system must temporarily have been in a similar state in those cases in which life has been restored after prolonged immersion for half an hour or more. In persons affected with the *morbus cœruleus*, when, from some malformation, the pulmonic and systemic circulations are no longer kept distinct, we have the best opportunities for observing the effect of an unnaturally venous condition of the blood. The following excellent description is given by Dr. Williams:—" Individuals thus affected are in a lower scale of animation. The slower processes of nutrition and secretion seem to go on pretty well, but the muscular power is low, slight exertions bring on symptoms of faintness, palpitation, suffocation, or insensibility, the animal heat is lower than natural, and there is greater suffering from the influence of cold. In short, all the powers of body and mind are slender, and are easily disordered by any circumstances which tax their activity. In a few that reach mature age there is no sexual passion. . . . . . The subjects of cyanosis are said to be very liable to hæmorrhages, and when these occur spontaneously, or from accidental causes, it is very difficult to stop them. This must be ascribed to the deficiency of fibrine, which we have already found to occur where the changes of the blood by respiration are imperfect."

In what way does accumulation of carbonic acid in the blood prove fatal to life? We find, after death from asphyxia, the left side of the heart comparatively empty, and its cavities contracted, the right side gorged with blood, as well as the veins generally; the lungs are also distended, and gorged with dark blood. Now, it has been shown that the essential cause of the failure of the circulation is not paralysis of the heart, or of the brain, though these may have some influence, but arrest of the blood in the capillaries of the lungs.

How this arrest is produced we have not sufficient positive information to enable us certainly to explain, but we see that it is a phenomenon of the same class as that congestion which has been mentioned as often occurring when the function of a part is suddenly put a stop to; or, as Dr. Carpenter expresses it generally, " the performance of the normal reaction between the blood and the surrounding medium (whether this be air, water, or solid organized tissue) is a condition necessary to the regular movement of the blood through the extreme vessels." The correctness of this position is almost demonstrated by the following experiment of Dr. Reid. Having adapted an hæmadynamometer to systemic artery of an animal, and obstructed its respiration, he found that when non-oxygenated blood was beginning to circulate, as shown by the commencing supervention of insensibility, the column of mercury in the tube was raised, indicating, of course, an increased resistance to the onward flow through the capillaries.

In this instance the normal changes between the non-arterialized blood and the tissues could not take place, and, consequently, the blood could not freely pass through them. The converse of this experiment is presented to us in the effect of cold on parts that are exposed to it. The functions of the part are abolished, the circulation languishes, and at last ceases, the vessels remaining congested with venous blood, which is not carried onwards. Hence the blue or livid colour which the surface presents. In this case the vital power of the tissues seems to be paralyzed by the sedative influence of the cold; and, as a consequence, their nutrition and circulation are also brought to a stand. The arrest of the pulmonary circulation in asphyxia seems very analogous to the foregoing instance; the normal changes in the lung tissue having ceased, the blood is no longer able to traverse its capillary plexus freely, but stagnates there, and congests the part. Thus far we simply class together a number of similar phenomena, and educe from the circumstances common to them a kind of law, viz., that quoted from Dr. Carpenter. But Dr. Draper has brought forward a view, which is extremely plausible and beautiful, and appears to us likely to prove of the greatest value in physiology and pathology. It is founded on the statement "that if two liquids communicate with one another in a capillary tube, or in a porous or parenchymatous structure, and have for that tube or structure different chemical affinities, movement will ensue, that liquid which has the most energetic affinity will move with the greatest velocity, and may even drive the other liquid entirely before it." The essential idea appears to be this, that the oncoming liquid is attracted, particle by particle, to various points of the tissue which it traverses, that the attraction having taken place soon ceases, in consequence of an alteration being effected in the attracted fluid, and that then the particles of fluid, no longer retained or drawn to the part by attraction, are pushed on by fresh quantities of unaltered fluid, for which the tissue has attraction. Thus, in the systemic capillaries, the arterialized blood is attracted to the tissues, changed by the act of nutrition to venous, therewith loses its capacity of being attracted, and is driven on by more arterial blood coming up within the range of the tissue's attraction. In the same way we may conceive an attraction to subsist between the venous blood and the air in the cells of the lung, which will, of course, cease as soon as the change from venous to arterial blood has been accomplished. The effect of this nutrition force, a term which we prefer to Dr. Carpenter's "capillary force," is evidently to promote remarkably the free transit of the blood through a part, and there can be no doubt that the arrest or abolition of this force must tend materially to obstruct the circulation. Hence, in asphyxia, the attraction probably continuing some time, but the normal changes which liberate each particle from it not taking place, the blood continually arrives and stagnates in the pulmonary capillaries. We have dwelt the longer on this subject, because it appears to us of so much importance to recognize the principle that the nutrition of a part influences so materially the circulation of blood through it, and because we may often have occasion to refer to the view here enunciated. The poisonous influence of carbonic acid is well shown by the following comparative experiment of Rolando. He tied one of the bronchi in a tortoise, and found that the animal was not materially injured thereby, but when, instead of merely cutting off the access of air, he furnished a supply of carbonic acid to that lung, the other still receiving air, the animal died in a few hours.

Uræmic Poisoning.—When the action of the *kidneys* is arrested, or seriously interfered with in any way, their secretion products are no longer carried out of the system, but remain in and contaminate the blood. The effects produced by the blood thus poisoned are somewhat different, according as the secretion is more or less suddenly and completely suppressed. When the suppression occurs suddenly the acute form of uræmia, as it is called, manifests itself. Frerichs describes three varieties of this. In the first, after some pain of the head, giddiness, or vomiting, the patient soon sinks into deep stupor, from which in no long while he cannot be aroused. In the second epileptic convulsions suddenly appear, affecting the whole muscular system, and returning after occasional intermissions. The consciousness may remain unaffected. In the third form both convulsions and coma occur. Such cases constitute the disease which received a separate name, as *ischuria renalis,* but they probably belong to the same class as the acute anasarca, which occurs sometimes spontaneously, or after scarlet or typhus fever, the anatomical characteristic of which is great sanguine engorgement of the kidney. Uræmia, in its chronic form, appears at the close of Bright's disease very frequently. Frerichs describes it as coming on gradually and unperceived, occasioning dull headache or confused sensation, impairing the mental and bodily faculties, and producing some dulness and drowsiness. These symptoms may remit if the urinary secretion increases, or they may progress, and become more intense, the drowsiness deepening into stupor and coma. Vomiting is a frequent symptom in uræmia, and sometimes amaurosis, or disturbance of the hearing is observed. Diarrhœa sometimes takes place, and seems to avert the dangerous consequences of uræmia: it was a prominent symptom in the animals whose kidneys were extirpated by Prevost and Dumas, and in those similarly treated by Bernard and Barreswill; in the latter it was particularly observed that large quantities of ammoniacal fluid were poured out by the mucous membrane of the stomach and intestinal canal; while these continued the cerebral functions were unimpaired, but as soon as they ceased the symptoms of intoxication commenced. Inflammations of the serous membranes, especially the pleura and pericardium, are very commonly produced by uræmic poisoning in a less severe form. Cases of pericarditis of renal origin are nearly as frequent as those of rheumatic. The term uræmia seems to imply that the poisoning of the blood depends on the presence of urea, and such has long been the general belief, but numerous experiments and observations of late have done much to invalidate it. The quantity of urea in the blood and the intensity of the symptoms bear no proportion to each other; there may be much urea in the blood and no symptoms, and severe symptoms with little or no urea in the blood. Dr. Todd, Vauquelin, and others have injected urea into the veins of animals, or given it by the mouth, without producing any other effect than increasing very greatly the flow of urine. Still more, Frerichs has repeatedly injected human urine into the blood of animals without producing any ill effects. It seems clear then that it is not urea, nor any other constituents of the urine, that produce by their presence in the blood the symptoms of poisoning. It may be, however, some of their decomposition products; and Frerichs states that he has proved it to be the carbonate of ammonia, which is well known to result from altered urea. He has repeatedly demonstrated the presence of ammonia in the air expired by the sick, and by animals into whose veins urea had been injected after extirpation of the kidneys. Carbonate of ammonia, he says, can always be detected in the blood whenever uræmic symptoms exist, as well as usually traces of undestroyed urea. The very exact experiments of Kühne have, however, shown that ammonia may be quite absent from the blood of animals dying of uræmic poisoning, since no reaction was given by a test sufficiently delicate to detect one-millionth part of carbonate of ammonia. The presence of an excess of urea in the blood, after extirpation of the kidneys, or ligature of the ureters, has been called in question, especially in cases of the former operation. The discrepancy seems to depend upon the amount of vomiting, since by this means much urea is got rid of. In rabbits, which never vomit, both these operations produce a considerable accumulation of urea in the blood. Succinic acid has also been found by Meissner in the blood of uræmic animals.* It has therefore been suggested that "uræmic poisoning" is due to the presence in the blood of nitrogenous bodies more complex than urea, which would, if the kidneys were acting normally, be changed into urea. What is the exact cause of the inflammations of the serous membranes, which often prove fatal in renal degenerations, does not seem made out; it does not appear to be the presence of urea in the blood, as we have no evidence that this is capable of producing such effects. Frerichs thinks that the impoverished state of the blood is an adequate cause, but in this we can hardly agree. While speaking of urea as a substance abnormally present in the blood, it must not be left unnoticed that it is only its presence in anything like considerable quantity that is abnormal, since it has been clearly proved that a minute quantity exists in perfectly healthy blood of men and animals. The same is the case with another constituent of the urine, viz., uric acid, which exists naturally in small proportion in healthy blood, but accumulates therein from defect in the excreting functions of the kidney just before an attack of acute gout, and also in chronic. As it is deficient in the urine Dr. Garrod's conclusion seems just, that the chalk-like deposits appear to depend on an action in and round the joints vicarious of the uric acid excreting function of the kidneys. The well-known effects of the presence of this gouty matter (uric acid) in the blood, as the *malaise* and ill-health which precede the attack, the inflammation produced by its localization, and the occasional serious result of its transfer to more vital parts, illustrate exceedingly well the disturbing action of an excretory substance retained in the blood. Another nitrogenous substance which has been found in the blood is hippuric acid; but it is not known to have any pathological significance. Other substances abnormally present in the blood are hypoxanthine and gelatine, which have been found by Scherer in leuchæmia. The former substance is of normal occurrence in the spleen; and the latter occurs in that organ in the same disease, which is accompanied by its presence in the blood. Hence they are probably derived from the white corpuscles; especially as Bödeker has obtained gelatine from pus corpuscles.

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* Kühne, "Physiologische Chemie," p. 250.

Whether *bile*, when present in the blood, is the cause of disorders, we do not certainly know. Its gradual disappearance as it passes down the intestinal canal is considered by Liebig as a proof that it is absorbed; but Lehmann is unable to find any trace of it in the blood of the portal vein. It may, therefore, be decomposed, and not absorbed. Biliary pigment is often present in the blood in considerable quantity without occasioning much disturbance, but we cannot speak so positively with regard to the biliary acids. It has already been stated that these substances, when injected into the blood, have a peculiar action on the red corpuscles; but this has never been observed in jaundice. In that terrible affection of the liver called by Rokitansky acute yellow atrophy, in which the cells of the organ are completely destroyed, and the whole tissue deluged with yellow pigment, we are quite ignorant of the exciting cause of the convulsions and coma by which the disease commonly proves fatal. No chemical examination that we know of has yet been made of the blood in this disease; and all that can be said is, that it seems most probable that the poisonous matter which produces the cerebral symptoms is none of the constituents of the bile, since both of them, the pigment and the cholic acid, have been found in the blood when none of the symptoms of cerebral disturbance were present. It may be, perhaps, a decomposition product of the organic biliary acids. However, though we cannot point out what the *materies morbi* in this case is, there is no doubt that the phenomena in this affection are owing to the presence of *some* abnormal matter circulating with the blood.

In that state of system which Dr. Prout has distinguished by the name of the *oxalic acid diathesis*, it has been thought that oxalic acid, or some of its salts, must be present in the blood, and be the exciting cause of the various symptoms. It has, however, never yet been detected in the blood, though it has been in urine and several other of the fluids of the body. It is, according to recent views, chemically in close relation to urea, and its occurrence is therefore not surprising.

Morbid Poisons.—The above-mentioned substances, abnormally present in the blood, and producing disease, are tolerably well defined, but there are many others of whose nature we are ignorant, and which quite escape our means of observation. The principal of them are infectious principles of the so-called Exanthematous diseases, including continued fevers; syphilis belongs to the same category, and various cutaneous disorders, especially the squamous and vesicular. Variola, and its modification vaccinia, are the only instances in which we can at all pretend actually to exhibit the *materies morbi*, and to transfer it from one system to another; even in these cases the visible fluid is but the vehicle of the poison, for that is aëriform, and capable of being received through the channel of the lungs. The venom of deadly snakes, perhaps, may be an instance in which the matter inducing the morbid alterations in the blood of the bitten person is manifest and palpable; but even here we have no knowledge what the substance is which produces the septic effects. In the case, however, of deleterious gases, and of most poisons, the toxic agent is clearly known, and we can form some idea of its mode of operation. It would be quite beyond our province to attempt any detail of the various poisons and the effects they produce; we can only observe that they are all referable with tolerable accuracy to three heads, or to two of these combined, viz., (1) poisons which act as irritants, producing more or less irritation and inflammation of various organs; (2) poisons which act as sedatives, causing paralysis, more or less immediate and complete, of the nervous system; (3) septic poisons, which seem to annihilate the vital power, and induce rapid putrefaction of all the organic fluids and solids.

With regard to the action of poisons there are two fundamental ideas which it seems desirable briefly to refer to. One is, that when a minute portion of virus is introduced into the system, it appears to multiply itself immensely, as if it possessed the power of transforming healthy matter into its own noxious nature. Such a multiplication must take place when an unprotected person is inoculated with the matter of variola, the minute quantity of virus introduced reproduces similar properties in the contents of the numberless pustules which are formed all over the surface. The same is doubtless the case with all infectious diseases, and with syphilis. The conception now mentioned applies more particularly to certain irritant poisons; the second to those that are termed septic. When spongy platinum is placed in a mixture of oxygen and hydrogen gases, they quickly unite together and form water, the platinum itself undergoing no change. This is an example of what Berzelius named "catalytic action;" there are many similar known instances, and it is very probable that actions of this kind are by no means infrequent in the animal system. The solution of the food in stomach digestion is probably, in part, dependent on a catalytic action, or one of a somewhat similar kind, in which the peculiar organic matter called *pepsin* disposes the alimentary ingesta to undergo solution in the gastric acids. A minute quantity of the change-inducing substance is sufficient to cause the action to commence, and so it appears a minute quantity of virus is sufficient to induce septic changes in the blood with which it is mingled. The history of cases of death from the bite of venomous reptiles, of the most malignant fevers, especially scarlatina, and of the effects of the matter of glanders, shows that the essential and primary action of these poisons is to lower extremely the vital powers, and induce putrefactive changes in the organic fluids.

ANÆMIA, SPANÆMIA.

There can be little doubt that by excessive hæmorrhage, or exhausting discharges, the whole mass of circulating fluid in the vascular system can be considerably reduced; that is to say, the result of such losses is not only to impoverish the quality (as we know it does), but to diminish the quantity of the whole mass of blood. The term *anœmia*, signifying absence or deficiency of blood, is therefore correct, though if it were not so commonly received and employed, one might wish to substitute the term *oligœmia*. *Spanœmia* is the name proposed by Dr. Franz Simon to express a deteriorated quality of the blood (*σπανος*, poor), it almost always accompanies the state of oligæmia, or anæmia; both may, we are inclined to think, exist not unfrequently as the sole condition itself, the blood being of normal quantity, but impaired quality. We will here recapitulate shortly the changes which have been before detailed in the several constituents of anæmic and spanæmic blood. (1.) The red corpuscles are remarkably diminished, 127 being the average per 1,000, they have been known to sink as low as 27; they also appear to contain less hæmatine, being somewhat paler than those of healthy blood. (2.) The amount of white corpuscles does not appear to be altered; in some of our examinations they have been found as numerous as in healthy blood. (3.) The fibrine is quite unaffected; it was never found below the normal mean; and in cases where inflammation of some organ was present its quantity was notably increased. (4.) The solids of the serum have not been found specially altered. (5.) The quantity of water is more or less increased in proportion to the diminution of the globules; in the case before mentioned, where the globules were only 27 per 1,000, the water was 886.

The *causes* which produce anæmia and spanæmia are—(1.) Losses of blood, whether natural or artificial, the red globules being thus diminished, their place is supplied in great degree only by the absorption of water. (2.) Profuse discharges of watery, mucous, or albuminous fluids, such as occur in aggravated leucorrhœa, diarrhœa, or in cases of cauliflower excrescence. In these it seems as if the blood globules were melted down to supply the profuse drain, probably they perish, or are not reproduced from want of a proper nutrient fluid. (3.) Insufficient food; the effect of a greatly-improved diet increasing the amount of red corpuscles was very apparent in a case under our observation, in which iron had been previously administered, with some but not marked benefit, while on the improvement of the diet the amelioration was rapid. Too often, no doubt, this cause operates powerfully in inducing the anæmia so common among young females of the lower classes. (4.) Deprivation of fresh air and light; the effect of this can scarcely be over-estimated. Even the best food will not be converted into healthy blood if light and air are withheld, while a coarse and insufficient nutriment will not prevent a person from having a ruddy colour, if he be much in the open air. Of this we have frequent instances among our labouring population. (5.) An unhealthy condition of the blood, in consequence of which the existing blood globules are imperfectly nourished, and the development of new ones is hindered. Such is the cause of the anæmia in persons suffering from degeneration of the kidneys, from lead cachexia, the cancerous diathesis, perhaps the tubercular, and in some chlorotic cases. There can be no question that in many cases, as is well described by Dr. Williams, the anæmia is not the cause but the result of the amenorrhœa. The suppression of the natural evacuation leaves the blood in an unpurified state, which is unfavourable to the development of healthy hæmatine. In the same way rheumatism may prove a cause of anæmia, and in several of Andral's cases the globules at the first bleeding were found not to be below the ordinary average. Mental anxiety may probably also be considered as a cause of this kind.

Results of Anæmia.—Several instances have occurred in which the nutrition of the heart had suffered so much, and the organ become so debilitated, that sudden and fâtal syncope was the result. The possibility of this should always be borne in mind, in treating a case of severe anæmia, and the patient should be enjoined to avoid sudden efforts, and to remain as quiet as possible until some degree of strength and tone is restored. It is a question of much interest, but as yet we believe not determined, whether the increased action of the heart above described ever produces structural change. It would be thought likely that a weakened hollow organ, contracting repeatedly for a long time on the mass of blood poured into it, would be apt to yield somewhat to the outward pressure or resistance of the fluid, and thus become dilated; but it does not seem to have been shown that this actually takes place. Some degree of anasarcous swelling of the feet and ankles is not uncommon. When this occurs we may take it as a sign, on Andral's authority, that the albumen of the serum is diminished. Asthenia may advance to such a degree that it proves fatal, by a gradual failure of the vital powers, like the sinking at the close of diseases of exhaustion. The impoverishment of the blood may probably be the determining cause of the appearance of tuberculous or other cachectic diseases. The nutrition of some parts in the anæmic may be impaired to such an extent that ulcerations form spontaneously. The cornea would appear especially likely to suffer in this way from not being permeated with vessels. Such instances of morbid action are very important to notice, as being free from complication, and exhibiting, therefore, more clearly the essential nature of a process. Ulceration in this case is clearly not produced by inflammation. It is rather interesting to remark that the most lowly organized, and the least essential of all the tissues, viz., the adipose, suffers less from impaired nutrition in the anæmic state than any other. It is by no means uncommon to see persons, especially females, presenting a considerable amount of *embonpoint*, who are manifestly very deficient in healthy blood. This is the more easily comprehensible, as the fat vesicles really seem to be scarcely more than so many minute drops of exuded oil, included in homogeneous films of protein material.

Local or Partial Anæmia.—The foregoing history of anæmia and spanæmia manifestly relates to it as a *general* condition. It seems very doubtful how far there can exist such a condition as partial anæmia, if we recognize a deficiency of red corpuscles as an essential feature of this state. Of course the supply of blood to a part may be defective, in consequence of various causes; but this does not involve any alteration in the quality of the fluid transmitted to the part. However, using the term in the sense of merely deficient supply of blood, the consequences of such a state will be generally those of diminished nutrition, or, more properly, atrophy of the part, with more or less considerable impairment of its function. If the deprivation of blood be very great, mortification may be the result; this has occurred in some cases in which the main artery of a limb had been tied on account of aneurism, and the collateral circulation did not establish itself soon enough. Even in cases which have a more favourable issue, the immediate effect of cutting off the supply of blood is to occasion weakness, numbness, and reduction of the temperature; the muscles and nerves are, in a great measure, paralyzed, and the heat-producing process fails with that of nutrition. When the aorta of an animal is tied, its lower extremities, after a time, become as paralyzed as if its spinal cord had been divided. The causes of local or partial anæmia may be, (1) tumours of various kinds, situated so as to press upon and obstruct the main artery supplying the part; (2) disease, often atheromatous, of the coats of the vessel itself, leading to deposition of fibrinous coagula, and consequent obstruction of the channel; (3) spontaneous coagulation of the blood in an artery; (4) blocking up of a vessel by fibrinous flakes transported from a distance, perhaps from the valves of the heart; (5) there is no doubt that during life anæmia of a part may be produced by spasm of the muscular walls of the small arteries supplying it, either dependent on nervous action or possibly spontaneous. Such a condition will not be recognisable after death.

It may be observed that it is difficult in many cases to say positively whether anæmia of an internal organ exists; we cannot observe during healthy life what amount of blood, as indicated by its colour, it contains, and the changes in the distribution of blood which may ensue during the last hours of life, and after death, will greatly alter the natural appearance.

HYPERÆMIA.

Hyperæmia, the opposite condition to anæmia, implies, of course, an excessive quantity of blood. The term is commonly applied to accumulation of blood in a part, *i.e.*, to local or partial excess; while plethora (*πλῆθος*, a multitude) is that which is used to signify increase of the general mass. We will first consider plethora, or general hyperæmia, and afterwards partial.

Plethora.—The characters of marked plethora are strongly expressed, and easily discernible. The face is rather full and turgid, and presents a diffused redness, often of a slightly purplish tint; this is especially observable in the lips. The conjunctivæ are redder than natural, the expression of the eye sharp and ferrety. The pulse is full, and more or less strong. The temperature of the skin is inclined to be hot, and even in the most remote parts it is fully maintained. There is a tendency to headache, and not unfrequently there is some degree of drowsiness and disinclination to exertion. Persons in this state have good appetites, and digest their food well, the secretions all seem to go on naturally, and organic or vegetative life is in full vigour. On account of the increase in the quantity of blood, it is manifest that complete oxygenation of it must be more difficult; hence, on any exertion the breath is apt to be short, and the action of the heart labouring; hence, also, as Rokitansky observes, the blood always presents a certain degree of venosity, as if never thoroughly arterialized. A distinction has been drawn between two principal varieties of plethora, the sthenic and the asthenic. Generally, it may be said, that in the one the organic life and tone of all parts are exalted, in the other proportionately depressed, while in both the mass of blood is in excess. Heat of skin, frequency (not, however, great) of pulse, with fulness and hardness, keen sensibility, mental and bodily activity and energy, a tendency to gout, bilious attacks, and disease of sthenic type, characterize the first form of plethora. It is observed in the "young, the active, and those of sanguine temperament." In asthenic plethora the skin is cool, the extremities apt to become cold; the pulse is large, but without resistance; it is often slow, sometimes irregular. The venosity of the blood is marked; the lips are often of a livid tint. The contractility and tone of the muscles is deficient, the spirits depressed, the mental and bodily activity diminished. It is most often seen in the aged, in those who are exhausted by excesses or previous disease, "or in whom the excreting organs act imperfectly." The characters of the blood in sthenic plethora are—(1.) The increase and amplification of the entire mass. Of this we have no direct measure, but we may form a tolerable idea of the extent to which it takes place by observing the effect of blood-letting. As much as forty or fifty ounces may be drawn at once in some cases, without fainting being produced; and even this quantity has sometimes been exceeded. Dr. Watson mentions a case in which seventy-two ounces were withdrawn before the patient became faint. (2.) The red globules in plethoric blood are remarkably increased, while the fibrine rather inclines to be somewhat diminished, and the albumen of the serum undergoes little variation. The quantity of water being diminished in proportion to the increase of the red corpuscles, it follows that the coagulum formed after bleeding will be large, and will be surrounded by but little serum. The mass of corpuscles, in proportion to fibrine is so great, that the latter cannot contract to the degree it ordinarily does; and hence a larger amount of serum is retained within the clot. In sthenic plethora the coagulum is firm, as well as large; in asthenic its cohesion is diminished. (3.) A tendency to deficient arterialization may also be mentioned as a character of plethoric blood; perhaps we may connect this with a deficient production of fibrine, which, as we before stated, may with much probability be regarded as an oxydation product.

Causes of Plethora.—Among the *causes* of plethora, the first place, perhaps, is to be assigned to a special tendency innate in the system to form an undue quantity of blood; or, speaking more exactly, to a too rapid growth and multiplication of the red corpuscles. This, as it requires, so it may produce an increased quantity of liquor sanguinis, according to the principle that the demand induces a supply. When the tendency to form blood is considerable it will manifest itself, even in spite of circumstances that oppose it; but a similar tendency, in much less degree, will produce a most highly plethoric state, if favoured by an ample supply of rich food, and a sedentary life. Indeed these may have the same effects, even supposing no predisposition to plethora at all to exist. It is worth remarking, however, that they will not produce this result in all cases. In many it would be rather dyspepsia, or some cutaneous disorder, or a bilious attack. Most of the circumstances that promote a robust state of health, with the exception of exercise, are favourable to plethora; and, on the other hand, such as depress the general vigour, or induce diseases of debility, prevent its development.* Asthenic plethora is probably in most cases dependent upon an unhealthy state of the liquor sanguinis, occasioned by impaired action of some of the excretory glands, which itself may depend on some latent organic diseases of the same.

Consequences of Plethora.—The consequences of plethora have already been in part alluded to. They are generally such as result from over-distension of the vascular system. On account of its proximity to the heart, its delicate structure, and the large supply of blood it receives, it is not surprising that the brain should suffer from this cause more than most other organs. Rupture of some of its thin-walled vessels may take place, or the blood be poured out from numerous capillaries, though it is doubtful whether these results ever occur except there be also some disease of the vessels. The great capacity of the vascular system of the liver will cause it to be enlarged by the increased distending force of the blood-mass, more, in proportion, than many other organs. This will be especially the case if dilated hypertrophy of the right chambers exist, and the blood is thrown back on the venous side of the circulation. Again, in consequence of hepatic congestion, the tributaries of the portal vein will also be congested; and this seems more especially to affect the hæmorrhoidal plexus of veins, which become distended into the little tumours, well known as "piles," and often give rise to a salutary hæmorrhage. Another hæmorrhage, not unfrequent in plethoric persons, especially the young, is from the veins of the nose. This seems especially to give relief to cerebral congestion. Menorrhagia may also be dependent on, or at least greatly increased, by a plethoric state. The natural determination of blood at the catamenial periods will of course be often attended with a greater discharge, on account of the increased tension of the vascular system. Though we consider it at least doubtful whether the plethoric are more prone to inflammation than others, there is no doubt that when inflammation is set up in them it is more violent, and requires more active treatment. Dr. Copland states also that the severer forms of fever in the West Indies affect young and plethoric strangers, rather than older residents, the aged, and the weakly. It is not improbable that plethora may play some part in producing a varicose state of the veins, but it is very doubtful whether aneurismal disease is ever occasioned by it.

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* There is no doubt that the cessation of habitual discharges, or their arrest by art, especially when suddenly effected, and without any corresponding modification of the system, induces a dangerous plethora. This should never be forgotten, not only in treating persons who are manifestly of plethoric habit, but even those who seem and are really in a different state. It seems that the vascular system, after having been long insufficiently filled with blood, cannot bear the amount of distension immediately which in the state of health would only be natural to it.

Local or Partial Hyperæmia.—We must remark with regard to local hyperæmia, as we did with respect to local anæmia, that it differs from general hyperæmia or plethora, not only in the less extent to which it exists, but in not involving any qualitative alteration of the blood. Local hyperæmia, in fact, is not exactly local plethora; it simply implies that too much blood is accumulated in the vessels of a part, without taking any count of the nature of this blood. Hyperæmia of a part is a phenomenon which naturally attracts attention, and has been considered and commented on from the earliest times. It exists in the most various conditions, from that of increased vital power of the functional activity of an organ to that of cessation of all action in it, its death, and decomposition. One of the best examples of a physiological and natural hyperæmia is afforded by the female breast during the period of lactation; the vessels proceeding to it enlarge considerably, and it manifestly receives much more blood than at other times. So great is the flow of blood to the part that it not unfrequently happens, owing to a deficiency of secretory power, that the healthy hyperæmia becomes excessive, and a cause of inflammatory disease. The limits of physiological hyperæmia are pretty wide. It is often striking to observe how much more blood is contained in the vessels of a part that is actively employed than would be present there under ordinary circumstances. Indeed, there is much to lead one to the belief that it is not so much the amount of hyperæmia that determines the transition from the healthy to the morbid state, as the alteration of the vital condition of the tissues of the part. This view is borne out by the fact that artificial hyperæmia produced in one side of the head by section of the cervical sympathetic, an experiment which has been performed in numberless instances, does not, as a rule, or without some additional irritation, lead to actual inflammation or disease of the part. Some experimenters, as Schiff, have observed hypertrophy affecting the hair and skin, but even this is a rare and occasional phenomenon.

VARIETIES OF HYPERÆMIA.

In endeavouring to study the various conditions under which hyperæmia occurs, we shall consider, *first*, hyperæmia with diminished motion of the blood in the part; *second*, hyperæmia with increased motion, or, as they have been also called, *Passive* and *Active Hyperæmia.* The first of these states may be designated *congestion*—the second, *determination of blood.* This arrangement has the advantage of classing together several conditions in which hyperæmia is a prominent phenomenon; but it is not certain—especially as respects determination of blood—that it is founded on that which is the essential circumstance in these two conditions. This, at present, is not possible, from the imperfection of our knowledge; but it may be well to bear in mind the above caution.

Passive Hyperœmia, or Congestion.—Congestion—employing the term to signify excess of blood in a part with diminished motion—affects chiefly the small veins of the part, and the capillaries that communicate with them. Hence its colour is inclined to be of a dark venous tint, unlike the more vivid blush of inflammatory redness. The part often exhibits patchy, irregularly-distended vessels, which can be emptied by pressure, but gradually fill again. Its temperature is not much, if at all, increased; and the pain felt in it is rather aching or dull than acute. The degree of swelling varies according to the cause producing the congestion and other circumstances; generally it is not very great. Congestion may exist alone, but often there are present also some exudations, the results either of it or of the condition which gave rise to it.

Causes of Congestion.—The two principal causes which produce congestion are—(1.) Obstructions of various kinds to the return of blood through the veins. (2.) A relaxed and toneless state of the capillaries and small veins. Of the first cause we have a good example in tying up the arm for venesection; the current of blood setting towards the heart being obstructed, and the artery continuing still to pour in fresh quantities, the capillaries and all the veins up to the obstructed part become distended with blood. This is marked by the red or purple colour of the part, and its swollen condition. The same effects will of course be produced in all instances where the veins of any organ are obstructed; obstruction of the jugular veins produces congestion of the brain, of the renal veins, congestion of the kidney, and so on. The modes in which the obstruction may be produced are very various; to take the brain as an instance, the veins returning the blood from it may be pressed on by an enlarged thyroid, or by a mass of indurated glands, or by an aneurismal tumour. Temporary cerebral congestion may also be caused by a prolonged expiration, or by holding the breath, especially when muscular exertion is made at the same time. The arrest of the blood in the veins in these cases depends partly on the diminution of the capacity of the chest, which takes place during expiration, partly on the suspension of the respiratory movements, which cause the blood as well as the air to rush into their respective cavities within the thorax during inspiration. Perhaps the most marked instance of the effects of this arrest is manifested by severe cases of hooping-cough. The capacity of the chest is narrowed more and more by the repeated expirations, and the blood not being drawn onwards, but thrown back, accumulates visibly in the face, which becomes turgid; in the eyes, where ecchymosis sometimes takes place; and similarly, though we cannot see it, in the brain, where extravasation may also occur, or such congestion as produces an attack of convulsions. Obstructive valvular disease of the heart, throwing the blood back on the lungs, is the cause of the abiding dyspnœa which characterizes such complaints. The condition of the large hepatic veins, and of the infr. cava where it receives them, shows that the influence of inspiration must be felt as a powerful cause in promoting the circulation through the liver; when this, therefore, is impaired, as is the case in vesicular emphysema, or other diseases interfering with the respiratory movements, the liver will be congested, and the same will of course occur when in consequence of asphyxiating causes the blood does not pass freely through the lungs, but accumulates in the right side of the heart, and in the large veins. Now, in all such instances of congestion it may be remarked that there is no evidence of excitement of the part, if we except, at least, the occurrence of convulsions in attacks of paroxysmal cough; the temperature of the part is not raised, the functional activity is rather diminished than increased, and exudations from the blood-vessels, if they occur, show no tendency to organization. Everything indicates that the hyperæmia is merely the result of a mechanical cause, and that there is no primary and special alteration of the vital endowments of the part.

Relaxation of the Vessels.—The second cause of congestion, viz., atony of the vessels, may occur either primarily or secondarily. In adynamic fevers, in states of extreme debility, and perhaps in some persons whose tonicity is naturally defective, the vessels of a part become distended with blood, without any obstruction existing in the veins which convey their blood away, or without any previous inflammation or undue excitement having exhausted their natural contractility. The whole surface of the body, in some fevers of very low type, is covered with patches of congested vessels, and it is to be noticed that these are chiefly seated in the under parts, the blood gravitating downwards, and accumulating in this situation. Hence we derive a hint for a precaution well worth observing in continued fever, viz., to alter the position of the patient occasionally, and not to allow the blood to gravitate day after day to the posterior parts of the lungs, which in consequence are especially prone to engorgement and hepatization. Primary atony of the vessels of the choroid coat of the eye seems not unfrequently to occur, and to be the cause of the muscæ volitantes to which the dyspeptic and others are subject. The vessels of the uterus in passive menorrhagia, and those of the vagina in non-inflammatory leucorrhœa, are not unfrequently affected by primary atonic congestion. It is, however, much more common that atony of the vessels occurs secondarily in consequence of some previous excitement or inflammation. This in fact is almost always the case in persons of feeble power, when they are attacked by inflammation; when the disease is subdued there still remains behind this congested state of the overstrained vessels, which being naturally of weak tonicity, are unable to resume readily their proper calibre. This condition of atonic congestion is often seen in the conjunctiva after it has suffered an attack of acute inflammation; and we can scarce have a more valuable lesson than the observation of such cases, and of the *lædentia* and *juvantia* offered us. A case of this kind is related in Mr. Tyrrell's work, on "Diseases of the Eye," vol. i. page 24, which from the first time we read it never passed from our mind; and often has the valuable instruction it conveyed been the means of directing us to successful treatment. Again, besides actual inflammation over-use of a part may occasion relaxation and congestion of its vessels. Of this we have an instance in the congestion of the choroid, which is so common in those who exert their eyes very much upon minute objects. Also in the extravasations met with in fatal cases of tetanus and chorea, in the brain and spinal cord, of which the *rationale* seems to be that the exhausted state of the nervous tissue has involved the minute vessels, and destroyed their retentive power.

Additional Causes of Congestion.—The influence of cold in producing congestions of internal organs cannot be doubted. To this we must ascribe, in part, the prevalence of chest affections during the colder part of the year, the blood being repelled inward from the surface by the constringing effect of the cold upon the vessels. The same cause also must be the chief agent in occasioning attacks of apoplexy, which have been observed to be greatly more frequent during a very cold season than during a mild one. The effect of the malarious poison in producing congestions of the internal organs is still more potent than that of cold. With every paroxysm the liver and spleen become greatly distended during the cold stage; and to such an extent may this take place that the latter organ, extensible as it is, has been ruptured, and fatal hæmorrhage ensued. Posture is a very efficient cause in producing congestion. It has already been alluded to, when instancing the pulmonary congestions that occur in fever; but the most marked examples of its effects are seen in the lower limbs. Here the returning venous current has to overcome the force of gravity, and though while the vessels maintain their tonicity, and the valves of the veins are efficient, this retarding force does not produce any effect, yet when the conditions are altered it becomes speedily manifest. Persons of feeble constitution, who are obliged to remain for the greater part of their time in an upright position, labouring hard, and living poorly, are exceedingly liable to a congested, thickened, and indurated state of the integuments of the lower part of the legs and feet. The veins proceeding from these parts—and especially the large superficial veins of the limb—are seen tortuous, enlarged, distended, and varicose. They are evidently gorged with slowly-moving blood, the column of which, greatly enlarged in bulk, has much more difficulty in resisting the force of gravity than in the natural state. Such congestions proceed very soon to ulceration, which is apt to assume a sloughing form, and which can only be healed by means which bring about a more healthy circulation. These are, of course, directed to take off the force of gravity by the recumbent posture, to empty the distended vessels, and to supply, by external equable pressure, the defective tonicity of their walls. It may, perhaps, be questioned whether the class of congestions from this cause should not have been included under those arising from venous obstruction, as the condition of the vein itself is such as to create an obstruction to its own current. Withdrawal of nervous influence from a part is sometimes the cause of congestion taking place in it. Mr. Simon records a case in which, after the ulnar nerve had been torn across at the inner condyle, the two inner fingers of the hand of the same side "had become swollen and livid with vascular injection." It is not, however, to be concluded that the walls of the vessels lose their tonicity whenever the nerves of a part are unable to discharge their functions, or that this is actually dependent on nervous influence. No doubt it is the special endowment of the vascular membranes, and only capable of being affected by the action of the nerves, as well as by direct stimulus. The effect of an atonic state of the walls of the vessels is well illustrated by an experiment performed by Dr. Williams; he adapted to a syringe a tube with two arms, one of which was connected with a metal tube, and the other with a portion of dog's intestine, of the same length as the metal tube; but, when distended, double its diameter. Water was now thrown in by the syringe, and the quantity discharged from the open ends of each of the two tubes estimated. The metal tube in the same time yielded three times more liquid than the intestine. Now a vessel, whose wall is possessed of a proper degree of contractility, may be compared to the metal tube. The force of the heart communicated by the fluid to its walls is not lost, but reacts again immediately upon the fluid, and drives it onward. The vessel with atonic flaccid walls resembles the intestine, which yielded to the distending force of the column of fluid, and, from not reacting upon it, allowed a great part of the force to be lost as an impelling influence.

Consequences of Congestion.—The effects of congestion have already been partially noticed. They have reference mainly to two circumstances—one the impairment of the vital actions of the part, the other the effusion from the overloaded vessels of watery, albuminous, or mucous fluids. When the arm is tied up for venesection a sensation of numbness, weakness, and chilliness is felt after a time, showing that the sensibility and contractility of the limb are impaired by the congestion which has taken place. In the hepatic and renal congestions, which often are produced by obstructive disease of the heart, the secretion of bile and of urine is commonly diminished, or morbidly affected. Cerebral congestion interferes materially with the free exercise of the functions of which the brain is the instrument.

This impairment of vital (*i.e.* special) power depends partly on the increased quantity of blood in the part, which, being greater than is proportionate to its functional activity, overloads and oppresses it. But a still more powerful, and quite unquestionable, cause of vital depression in a congested part, is afforded by the altered condition of the blood itself, which, semi-stagnating in the capillaries and veins, become more venous than it should, and otherwise unfit for the healthy nutrition of the tissues. The sloughing ulcers which form in the congested and thickened integuments of the lower limbs, when the veins are enlarged and varicose, are a striking instance of the lowered condition of the vitality of those textures. Long-continued congestion of the liver, from disease of the heart, produces a very remarkable effect on the parenchymal cells. They become very greatly loaded with yellow matter (which does not appear to be true biliary, but rather bile pigment); in extreme cases, the majority become atrophied and are reduced to a mere granular detritus, while the intercellular capillaries become enlarged. These changes afford some explanation why the secretion of bile is interfered with, and also why jaundice occurs.

The effusions that take place from congested parts are certainly the most prominent phenomena of the condition. They will be most abundant, as a general rule, when the congestion depends on venous obstruction; so that while fresh blood is being poured into the part no exit can be found for it, except that which the exudation affords. They may also be extremely abundant in some cases, in which not only the tonicity of the vessels is entirely lost, but the texture of their walls is altered, so that they no longer oppose any obstacle to the escape of their contents, but allow them (the fluid part) to transude with great facility. Such is, no doubt, the case in instances of bronchorrhœa, chronic diarrhœa, and leucorrhœa, where large quantities of fluid are continually passing off from the toneless vessels, but where no venous obstruction exists, or none that is commensurate to account for the discharge. The effect of remedies in these cases shows that the discharge is dependent on the cause we have mentioned. Under the administration of turpentine or astringents the vessels regain their tone, and no longer pour out their contents. Though the vascular atony may in such states have originated in congestion, yet as this is removed by the effusion while the atony remains, they come at last to be rather instances of passive flux, or dropsy. Chronic ascites, in which the smaller branches of the portal vein may be so obstructed that no fluid can pass through them, presents an exquisite instance of effusion depending upon venous obstruction. The fluid effused in the peritoneal sac varies a good deal, chiefly as to the relative proportions of water and albumen which it contains. Not only does a more or less watery serum exude, but fibrine not unfrequently accompanies it. Large flaky masses of fibrinous coagula are not uncommonly found in the peritoneal cavity after death, in cases of ascites, and the same are also seen occasionally in the fluid evacuated by paracentesis. We have also seen blood-globules so uniformly dispersed through the fluid that there could be no doubt that they had escaped from the congested sub-serous capillaries, and were not accidentally mingled with the effusion. The same products of congestion also occur in the urine when obstructive disease of the heart throws back the blood on the renal veins. The secretion is albuminous, contains fibrinous casts of the tubes and blood-globules. Decided hæmorrhage may also occur, as the result of extreme congestion, which may be dependent either upon venous obstruction or upon an atonic state of the vessels. Melæna, or hæmorrhage from the bowels, is an instance of the first, passive menorrhagia and epistaxis of the latter.

Textural Changes resulting from Congestion.—It is a remarkable and instructive fact that congestion of parts never seems in the end to occasion hypertrophy; or, if this should appear to have taken place, closer examination proves that rather the reverse is the case; that the hypertrophy is what Rokitansky calls *unreal.* Enlargement of organs may indeed be a consequence of venous congestion, if it has not continued long enough seriously to interfere with their nutrition; and is especially seen in very vascular parts, where the mere dilatation of capillaries and veins is doubtless one of the main causes of enlargement. The spleen, for instance, is usually found enlarged in cases of cirrhosis of the liver, where the portal vein is obstructed; and obstructive disease of the heart produces at a certain stage of the disease enlargement of the liver and kidneys. In the latter cases, however, certainly, and probably in the first the enlargement is only temporary, and the impaired nutrition which results from an inadequate supply of proper blood leads to a condition of atrophy, the external appearance of which will vary according to the organ involved. In the skin there will be ulceration; in solid organs, such as the liver and kidney, there will be what is called fibroid degeneration, or better fibroid substitution; that is to say, a replacement of the proper secreting structure by a certain form of connective tissue; so that the organ will be in most cases actually smaller; at all events wasted, so far as its proper structure is concerned.

We have already pointed out the impairment of vital power which congestion occasions, and the causes inducing it, which seem to afford an adequate explanation of the non-tendency to growth and development which is observed both in the affected part itself and in its interstitial effusions. The following sentence from Mr. Simon's lecture recognizes and similarly explains the same fact: "It is true that much blood is contained in the affected tissue; but it is blood that has insufficient means of renewing itself; and from its long detention in the part it acquires in an extreme degree, the character of venous blood. Thus, as regards mere bulk of blood, the part is over-supplied. but, in respect of the quality of blood, it may be said to suffer what is equivalent to anæmia. . . . . I think it not improbable that the same fact may contribute to explain the continued non-development of those effusions which arise from passive hyperæmia." It thus appears that atrophy, rather than hypertrophy, is likely to be the result of abiding congestion of any part.

Remote Consequences of Congestion.—The effects of congestion of a part, especially if it be one of some magnitude, may not be confined to the part itself, but may affect the system generally. This appears in two respects, one being a degree of faintness and depression (v. Junod's boot), occasioned by the withdrawal of a considerable quantity of blood from active circulation; the other, an injurious influence exercised on the whole blood-mass by the deteriorated portion, which slowly and partially returns into it again from the seat of congestion. It is clear that an excess of blood poured into one part, and detained there, must leave others imperfectly supplied; the local hyperæmia, according to its extent, produces a degree of general anæmia. That blood which has long been stagnant in a part must be in an unhealthy state is very comprehensible, and that, by its mingling with the general mass of blood, deterioration of the same will be induced, and therewith a cachectic state.

ACTIVE HYPERÆMIA—DETERMINATION OF BLOOD.

In this, the second variety of local hyperæmia, the phenomena convey the idea of increased activity and vigour in the vital process. The flow of blood to the part is increased; the capillaries, without being greatly distended, are well filled, and the blood which they contain having an arterial rather than a venous character, gives a red flush to the part, very different from the duller tint of congestion. In consequence of the fullness of the capillaries, and perhaps also of some increased exudation, there is some turgescence of the parts, and its temperature is elevated. The arteries also become enlarged, to admit the increased quantity of blood, and pulsate with more than ordinary force. This enlargement becomes permanent when the hyperæmia is a permanent and natural state. The veins also enlarge, but are not distended, as in congestion. The sensibility of the part is commonly increased; its function may be, or may not, and the same is true of its growth.

Distinction of Healthy and Morbid Hyperæmia.—This brings us to the inquiry whether active hyperæmia may not be distinguished into two forms, one to be regarded as healthy, associated with increased vital power and capacity for action; the other morbid, attending upon and promoting unnatural action, resulting in disease and decay There are many well-known and oft-quoted examples of healthy hyperæmia, such as the female breasts during gestation and lactation, the uterus during the period of pregnancy, the gums during dentition, the mucous membrane of the stomach while the secretion of gastric juice is going on, and, generally, it may be said, every organ during the time of increased activity and employment. Now, we think it may be affirmed, that, in the above instances, *the most important and characteristic phenomenon is the increased functional energy and vigour* of the hyperæmic part; this we believe to be the main and essential circumstance of which the increased blood-flow is a sequel. Nay, there are many instances, especially among secretory organs, in which the great increase of the product proves that a corresponding increase of the supply of blood must have taken place; a true hyperæmia, in one sense, exists, but it is not apparent, because of the active transformation which is going on. How marked is the difference between this condition and congestion! In the one, functional activity and molecular change at its height, with vascular injection more or less considerable, but not varying in direct, rather in inverse ratio to it; in the other, vascular injection extreme, while the functional activity is extremely depressed. Now, in proportion as active hyperæmia departs from the physiological condition, and becomes morbid—that is to say, approaches towards inflammation—in the same degree does the vital energy and activity of the part appear to be lowered, and the hyperæmia becomes the more marked phenomenon. Thus a diuretic drug shall be administered to two individuals; in the one the flow of urine shall be considerably augmented, the vital power of the kidney predominating over the hyperæmia excited; in the other, the flow of urine shall be diminished, and the secretion become bloody and albuminous, evidencing the predominance of the hyperæmia over the vital power. So, too, in fever. The skin, at one time, shall be dry and burning and red, with vascular injection, but its vital power of secretion and exhalation is in abeyance. But a change comes, the tissue regains its functions, and pours out a healthy moisture on the surface; and now the hyperæmia, though it may continue in some degree, is no longer predominant. How different must be the condition of the gastric mucous membrane in the hyperæmia excited by a few grains of ginger, and that produced by a few grains of arsenic! No doubt the one form of active hyperæmia may pass into the other. The quantity of blood which a healthy tissue was able to employ, and which was requisite for the unusually vigorous discharge of its function, may become too much for the same tissue when debilitated by over-use. A brain ministering to an active mind requires and receives a greater supply of blood than that of the waterman "who rows along thinking of nothing at all." So long as the cerebral energy is not over-tasked, the hyperæmia will tend to no injurious result, but will only supply the necessary pabulum for the material changes connected with thought. When, however, the time arrives that the delicate organ needs repose, then if the strain be continued, and the hyperæmia kept up, it is manifest that a morbid state will soon supervene, in which the hyperæmia may yet further increase, and the natural energy be still more diminished, till, together with symptoms of disordered and erring action, inflammation, or some other result of hyperæmia occurs. The phenomena we have alluded to are of every-day occurrence. No doubt can exist about their reality, though different opinions may be entertained as to how they should be interpreted. We shall immediately proceed to consider more closely the different views that have been advanced, but we would ask especial attention to this point which we have dwelt on, viz., that in one form of active hyperæmia the vigour of the tissues for vital action is increased, while, in the other, it is diminished. In the one the hyperæmia supplies a want, in the other imposes a burden.

Conditions of Normal Circulation.—Now, before we enter on the consideration of the mode in which active hyperæmia is brought about, let us refer shortly to one or two physiological points, which must form the very basis of all our attempts at explanation of the phenomena. All tissues may be regarded as consisting of vessels, nerves, and the proper elements of the tissue. The vessels, it is true, may be more or less closely woven up with the tissue, or even may not actually penetrate it, but still they are essential. The nerves convey an influence which may affect the blood-vessels or the tissue. The elements of the tissue, be they cells, or originally derived from cells, have certain special endowments, which, when called into action, increase the flow of blood to the part; at least this seems only another form of putting the undoubted fact—that exercise of a part causes more blood to flow thither. The blood-vessels consist of arteries, capillaries, and veins. The arteries have a truly contractile coat, which, under some kinds of stimulation, may even produce obliteration of the channel. This contractile coat is composed of organic muscular fibre. It possesses, so far as we know, no other property than that of contracting. There is not the least reason to suppose that it has any power of active dilatation.* The capillaries have a simple, homogeneous, membranous wall, in contact with the tissue proper of the parts. It does not appear that this possesses any contractility. It does, however, possess a retentive power, which varies remarkably in different organs—compare, for instance, the renal, the pulmonary, and the muscular capillaries—and is capable of being greatly impaired by morbid influences. The contractility of the veins has generally been considered much more doubtful than that of the arteries. Weber did not find them respond to the stimulus of cold as the arteries did. Mr. Wharton Jones, admitting that they do undergo some variation in size, says that the contraction or dilatation is very small in comparison with that which the arteries undergo. Professor Paget's observation seems conclusive as the contraction of both veins and arteries under a mechanical stimulus. He says, "If, as one is watching the movement of blood in a companion-artery and vein, the point of a fine needle be drawn across them three or four times, without apparently injuring them, or the membrane over them, they will both presently gradually contract and close; then after holding themselves in the contracted state for a few minutes, they will begin again to open, and, gradually dilating, will acquire a larger size than they had before the stimulus was applied." It must be noticed that Mr. Paget's observation has reference to the wing of the bat, while Mr. Wharton Jones' conclusion has been formed from examination of the web of the frog. The former, as a warm-blooded mammal, is no doubt a better representative of man than the reptile.

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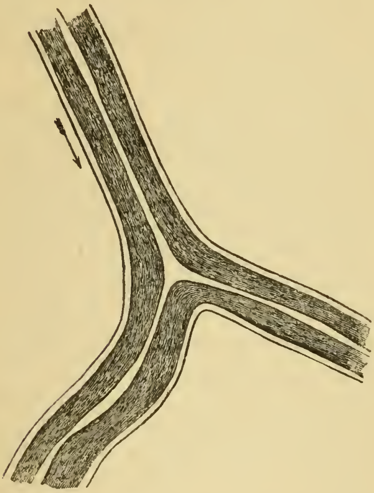
* Drs. Pettigrew and Schiff contend, however, for the affirmative.

Fig. 3.



Contracted artery, from Wharton Jones' Essay.

Phenomena of Irritation.—What we can see, by the microscope, in parts that are the seat of active hyperæmia, in consequence of the application of a stimulus, is by the very accordant testimony of the best observers as follows: — When a moderate irritant, such as tincture of capsicum, or a drop of some essential oil, is applied to a transparent part the arteries speedily dilate, and a rapid flow of blood through them ensues. This seems to tell on the capillaries and veins, which become dilated also, so that vessels, which before scarcely admitted blood globules, are now traversed by great numbers. Sometimes the dilatation seems to be preceded by constriction of the artery; but this does not constantly occur; and when it does is of very brief duration. It has been often said that the flow of blood was *accelerated* in arteries that were contracted; but the reverse seems rather to be the truth. It is the *dilatation* of an artery that is accompanied by greater rapidity of the current, though it cannot be the cause of it. Professor Paget writes, "As the vessels are contracting the blood flows in them more slowly, or begins to oscillate; nay, sometimes, even before the vessels begin visibly to contract, one may observe that the blood moves more slowly in them, as if this were the first effect of the stimulus. Nor am I sure that I have ever seen (what is commonly described) the acceleration of the flow of blood in the contracting vessels. Such an acceleration, however, is manifest, as the vessels re-open; and as they dilate, so apparently in the same proportion does the flow of blood through them become more free, till at length it is quite manifest that they are traversed by both fuller and more rapid streams than passed through them before the stimulus was applied." Mr. Wharton Jones' observation is to the same effect: "In one case," he says, "the arteries of the web were more or less constricted, the circulation sluggish, the blood in the capillaries here and there stagnant. A drop of the solution of sulphate of copper with vin. opii was applied, whereupon the arteries immediately became dilated, and the circulation brisk." Dr. Williams, in his work, so often referred to, had previously maintained and clearly illustrated the same view. It may therefore be considered established that in active hyperæmia one principal feature is dilatation of the arteries; while in passive hyperæmia the veins and the capillaries opening into them are dilated, and the arteries either are not enlarged, or are constricted.

Neuropathological Theory.—Now, it may very naturally be asked, in what way does the stimulus applied bring about dilatation of the arteries? All that we know of the habits of contractile tissues leads us to believe that they can only respond to a stimulus by exerting their contracting power, and there is no known instance of active elongation. We know indeed, from the researches on the influence of nerves over vessels, which started with Claude Bernard, that division of the sympathetic branch supplying any vessel, produces a paralysis of the vascular muscles which permits their easier dilatation, and hence a more rapid flow of blood to the part; and Bernard himself also believes in an active dilatation of the vessels as a consequence of a peculiar stimulation of the vasomotor nerves, though this is not generally accepted. It has also been shown that the same effects on the vessels may be produced by reflex nervous action, a fact very important for our present purpose. The theory was long ago proposed that the stimulus, acting on the sensory nerves of the part, excited in them a state, which, being communicated to the vasomotor centre, was reflected on the vascular nerves, occasioning them to become paralyzed, and therewith the contractile coat of the vessels also. This theory (the neuro-pathological) has lately received much support from the physiological experiments of Ludwig and others, and is accepted without hesitation by Dr. Burdon Sanderson.* When under ordinary circumstances a stimulus applied to a part produces increased activity of the capillary circulation, it is impossible to distinguish between the effects of irritation of the tissues themselves and irritation applied to the extremities of the sensory nerves. If, however, a sensory nerve trunk be divided, and the central portion irritated by feeble induced currents of electricity, the same phenomena of active hyperæmia are observed. The congestion is similar to that produced by section of the sympathetic, but more intense, and is accompanied by obvious dilatation of the arteries, not always preceded by contraction.* General acceleration of the flow of blood through the capillaries has also been produced by the excitation in the same manner of a special nerve in the rabbit, called by Cyon and Ludwig the *depressor*—a branch of the vagus. All these effects appear to be reflex, and the action to pass through a part of the central nervous system, the medulla oblongata. According to the most recent researches, those of Owsjannikow, this "vasomotor centre" is in the rabbit situated immediately below the corpora quadrigemina, and above the calamus scriptorius.†

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* Holmes's "System of Surgery," vol. v.: article "Inflammation," p. 729.

The path taken by the reflected nervous stimulus must be through the sympathetic to the vasomotor nerves; and this consideration lessens the force of certain objections to this theory. For instance, it appears a strong *primâ facie* objection that, as we know, a paralyzed limb can be the seat of active hyperæmia, and that, as Mr. Simon has shown, "the absence of a spinal cord, or the division of all the roots of the nerves, or the section of the lumbar and sciatic plexus, will make little or no difference to the certainty with which an irritant, applied to the web of a frog's foot, will quicken the circulation there, and subsequently lead to its retardation and arrest."

It is impossible to deny that the neuro-pathological theory, in its present form, offers an adequate explanation of the phenomena of hyperæmia from irritation; but it is by no means proved that an external stimulus *always* acts in this indirect manner.

Primary Changes in the Tissues.—We are accordingly led to seek for an explanation in the direct action of stimuli on the elements of the tissues, without the intervention of nerves and muscles; the latter being, on this supposition, only secondarily affected, in consequence of the molecular changes which take place in the elements. We will therefore briefly put together the arguments which appear to us conclusive, that what we have called the "nutrition force," and Dr. Carpenter the "capillary," does really exist, and is concerned in producing the state of active hyperæmia. (1). When a part is not employed (a limb, for instance) for some time, it wastes and atrophies. Its blood-vessels become smaller, and its temperature falls. Manifestly the circulation of blood through it is diminished. (2). When a part (as a limb) is actively employed, it enlarges, its temperature is increased, its blood-vessels are more developed, and the quantity of blood passing through it is evidently greater. (3). When a gland is excited to increased action, as the mamma of the female, the flow of blood to it is increased, and the vessels become enlarged. (4). In plants it has been observed, among other instances of the influence of local stimuli, that a branch of a tree, growing in the open air, which is brought into the atmosphere of a hot-house, will vegetate during the winter, and draw up sap through the stems and roots, while the other branches remain in their ordinary state. (5). In many of the lower invertebrata, the movement of the nutrient fluid seems to be evidently independent of the action of a heart on the vessels. (6). Dr. Houston's case of an acardiac fœtus has proved, in the judgment of those most competent to decide,* "that a fœtus may grow to a considerable size, and have its various tissues well developed, without any connection with the twin fœtus, by means exclusively of a circulation of its own, of which a heart forms no portion, or upon which it can exercise but a very remote influence." (7). Though the phenomenon of blushing, and some other local determinations of blood, may be accounted for by an alteration taking place in the calibre of the blood-vessels, their channels being widened, and more blood admitted, yet that of hyperæmia, excited by a local stimulus, appears to us quite impossible to explain in such a way. We would refer more particularly to the interesting experiment, recorded by Mr. Simon (p. 96 of his "Lectures on Pathology"), in which hyperæmia was induced by the local application of a stimulus to a part which had lost all trace of sensibility. This appears to us to afford conclusive proof, that neither the action of the heart, nor that of the blood-vessels, but only the nutritive force, heightened by the action of the stimulus, could have produced the local erythema. This justly eminent authority adds, "Altogether we may, I think, take it as an established certainty, that the first change which occurs in an inflamed or overgrowing part, and which leads to its becoming loaded with blood, is not a reflex change operated through the nerves, but is a direct change, operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood." He compares it to "a vortex, established in the place of the irritant, causing all the adjoining streamlets of blood to converge in swifter channels towards it." Professor Paget says, "I think I can be quite sure that the velocity of the stream, in any vessel of an inflamed part, is not determined by the diminution or enlargement of the channel. Without change of size, the stream may be seen decreasing from extreme velocity to complete stagnation. On what the alteration of movement of the blood in such a case depends, I cannot tell; but we have facts enough to justify such an hypothesis as that there may be some mutual relation between the blood and its vessels, on the parts around them, which, being natural, permits the most easy transit of the blood, but, being disturbed, increases the hindrances to its passage."

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* Lovén, "Ludwig's Arbeiten aus der Physiologischen Anstalt zu Leipzig," 1866; pp. 1 to 26. Cyon and Ludwig, *ibid.*, pp. 128 to 149.

† Berichte der K. Sächs, Gesellschaft der Wissenschaften. Math. Phys. Classe. May 6th, 1871.

Other forms of Active Hyperœmia.—The foregoing remarks apply chiefly to active hyperæmia resulting from the application of a local stimulus. There are, however, other forms in which the starting-point of the change has to be sought away from the part affected. We must first, however, notice simple *removal of pressure* as an immediate and certain cause of active hyperæmia. One instance of this is the removal of atmospheric pressure from the skin, in the operation of cupping. This acts, doubtless, simply by diminishing resistance to the passage of the blood, the motive force remaining the same. Nervous influences may also diminish resistance by lowering the tension of the arterial muscles. Section of the sympathetic, as has been shown by innumerable experiments, produces an immediate relaxation of the vascular walls and dilatation of their channels. Section of other nerves of the cerebro-spinal system has the same effect, probably because they contain fibres (vasomotor) derived from the sympathetic. This has been seen, for instance, after injury or experimental division of the ulnar, or of the fifth cranial nerve; that is to say, both in sensori-motor, or mixed, and in purely sensory nerves. Another important form of local hyperæmia is compensatory or collateral hyperæmia. When an arterial branch is blocked up, the parts supplied by it are at first anæmic, from diminished supply of blood; but the collateral circulation, by means of anastomosing branches, soon becomes established. Small branches in the surrounding parts become dilated to a large size, capillaries become as large as arteries, and thus the parts surrounding the region immediately supplied by the obstructed arterial branch become hyperæmic. But the process may go farther still, and, as shown by Cohnheim, in consequence of regurgitation from the veins, the *precise territory supplied by the obstructed arterial branch may become hyperæmic.* This fact, which appears paradoxical, has now been clearly established by the experiments of Cohn, Feltz, Savory, Cohnheim, and others, as regards small arterial branches or capillaries. In the obstruction of large arteries, such as those of the limbs, it is not seen, the collateral circulation being inadequate. As, however, the blood remains stagnant, and is or becomes venous in character, the active hyperæmia is really passive hyperæmia, or congestion, and leads to other changes.

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* Todd and Bowman, Phys. Anat. vol. ii. p. 372.

Relations between the Blood and the Tissues.—While admitting the reality of all these causes, we must regard it as far too exclusive and onesided a view to consider only the blood and the vessels as the agents concerned in hyperæmia, the common initiatory step of inflammation, and to deny to the essential elements of the part any share in the production of a state by which they are so importantly affected. We therefore recognize an increased attraction of the blood towards the part which is stimulated, as one cause of active hyperæmia, and the principal, and we regard the dilatation of the arteries as a secondary, but not unimportant. But the influence which the tissues exert on the circulation, in virtue of their "nutritive power," we may be sure is not only an attraction which may be increased or diminished, but also an alteration which the attracted blood undergoes, and, having undergone, is either repelled or pushed on by the advancing current. We may illustrate this motive influence by the example of light bodies, when acted on by electricity. Two pith balls, one of which is in a negative and the other in a positive state, will attract each other strongly; but as soon as they both become negative or positive, they forcibly repel each other. Some similar relation must subsist between the blood and the tissues. The arterial blood is heterogeneous to the tissue, and is attracted to it. Having become venous, it is no longer so, and it ceases to be attracted, perhaps even is repelled. Now, we may conceive the attractive force to persist, or even to be exalted, while the change impressed in nutrition may be greatly lessened. Blood will then accumulate in the part, from not having undergone that vital change which it should, and the part will be hyperæmic. This would be the case in active hyperæmia of a morbid kind—in that which forms the first stage of common inflammation, in which the vital endowments of the part are lowered, and its functional activity lessened. In healthy hyperæmia, on the other hand, the attractive and the changing influences are both increased. The blood does not accumulate, but only ministers adequately to the increased functional activity of the part.

EFFECTS OF ACTIVE HYPERÆMIA.

These, as already intimated, are different in the healthy and morbid varieties. In the first, the growth of the part, if the hyperæmia continue long enough, is increased, it undergoes a true hypertrophy. Of this we have the best examples in the muscular tissue. At the same time, the function is more vigorously exercised; it has more capability for, and it performs more work. Of this the brain, under the influence of moderate determination of blood—such as some of our great orators used to induce by pretty free libations of wine—is a good example. Similar instances among glandular organs we have already noticed. That of the ovaries and uterus, at the catamenial periods, is very remarkable, and is evidently connected with the reproductive nisus, which manifests itself especially at these epochs. There seems no ground whatever to regard it as originated by nervous influence, but rather as the result of a mode of growth and nutrient action peculiar to these organs. The discharge of the ovarian ovum, and the catamenial flow, are the results of this hyperæmia, but it is itself excited by the spontaneous activity of the structures. Thus curiously, as we see also in many other instances, are linked together the increased action of an organ and the increase of its supply of blood; in the healthy state, the former usually takes the initiative, and produces the latter, but is itself reacted on by it, increased, and carried on.

But it is rather with morbid hyperæmia that we are concerned as pathologists. This is, in a very great number of instances, the commencement of inflammation; but we shall not speak of inflammation as one of its results, but consider it separately. The effects of morbid hyperæmia are generally unnatural excitement, or oppression of an organ. The part contains more blood than it is able to manage, its healthy play is interfered with, and it is either goaded into a false, aimless, and exhausting activity, or is actually oppressed and enfeebled directly. The chief features of the condition are, probably, increased attraction of blood to the part, with diminished vital change, and undue dilatation of the arteries leading to it.

If the brain be the seat of determination of blood, in a morbid sense, there will be throbbing of the carotids, and their superficial branches, restlessness, more or less intolerance of light and sound, diminution of the power of attention and application, dreamy and disturbed sleep, irritability of temper, attacks of giddiness, &c. The face and eyes are apt to be flushed, and the feet cold. The uneasy sensations about the head are increased by stooping, or the recumbent posture. The kidney, in cases of acute anasarca, manifests an excellent example of morbid hyperæmia. It is enlarged and turgid with blood, but its texture is not apparently altered. Its secretion is scanty, loaded with albumen and with fibrinous concretions, and epithelium of the tubes. No doubt can exist that its functional energy is gravely impaired. In active menorrhagia we have a third instance in which, from various causes, a morbid hyperæmia of a hollow organ lined by a secreting mucous membrance is induced, the results being pain and uneasiness in the region of the affected part, increased sensation of heat, tension, and throbbing, which are relieved by the discharge of a fluid more completely sanguineous than the natural secretion; in fact, by an almost real hæmorrhage. In such an instance it is not only the mucous lining of the uterus that becomes hyperæmic, but the whole organ, with its thick muscular walls. Their tissue is loosened up and swollen by the quantity of blood admitted, so that the size of the organ is increased; and if this hyperæmia should not in great degree subside, the result may be a permanent enlargement and congested state of the uterus. The foregoing examples, taken from different organs, will serve as sufficient illustrations of the effects of active hyperæmia. It seems, however, desirable to allude, somewhat more in detail than has yet been done, to *hæmorrhage*, *flux*, and *dropsy*, considered as results of hyperæmia in general.

These may be regarded as the effusions of hyperæmia, as distinguished from inflammation. No doubt they do also occur in cases where inflammatory action is proceeding; but still they are not the special and characteristic products of this state.

HÆMORRHAGE.

This term implies the effusion of blood in mass, not merely of some of its constituents; exudations, therefore, which are only coloured by hæmatine, do not constitute hæmorrhage. The best character of an hæmorrhagic effusion is the presence of large masses of blood-globules imbedded in fibrinous coagula. Such may proceed either from an opening in a vessel of some magnitude, or from numerous capillaries. In every case where blood is effused in large quantity, the walls of the vessels must have given way; and perhaps this is the case in every instance where a blood-globule escapes from its channel, though it is not, to our minds, absolutely certain that there is no such thing as the hæmorrhage by exhalation of the older writers. This suspicion, expressed some seventeen years ago, has been converted into a certainty by the researches of late years. It is not now doubted that both red and white corpuscles can leave the blood-vessels without any rupture of their wall. Opinions are, however, still divided as to the precise manner in which this occurs; some think (with Stricker) that the vascular wall takes the blood-corpuscle into its substance, and discharges it on the other side; while Cohnheim believes in pre-existing openings in the walls, which are enlarged when the vessel is dilated, and allow the corpuscles to pass. This will be more specially considered under the head of inflammation; but there are reasons for thinking that the same thing happens without inflammation, if not under normal conditions.

Hæmorrhage may take place either in solid parenchymatous organs, or in those that enclose cavities and form canals. In the first case, the substance of the organ undergoes more injury than in the latter. An effusion of blood into the brain is a most serious thing; on the surface of the Schneiderian membrane it is a mere trifle. When a large quantity of blood is suddenly extravasated in a solid organ, it ploughs up and disorganizes the tissue, and forms therein a cavity for itself, where it lies like a mass of black currant jelly; the walls of the cavity are usually ragged, and soon become stained to some depth by altered colouring matter. But the extravasation may occur in a very different manner, affecting a great number of points at once, and having the appearance of a multitude of red dots scattered about, or of minute streaks. This is termed capillary apoplexy, to distinguish it from the other form ("apoplect. heerd" of Rokitansky). It is often seen in the grey matter of the cerebral hemispheres after death from concussion of the brain. If the hæmorrhage, though taking place in the same way, be more abundant, the spots and streaks approach closer together, the tissue becomes more swollen, and, at last, may become thoroughly red, the blood having penetrated completely between and among the elements of the parts. This is hæmorrhagic infiltration, and when occurring in a limited region, all parts of which outside the vessels become occupied with blood (as the alveoli in the lungs), it is called a hæmorrhagic infarctus.

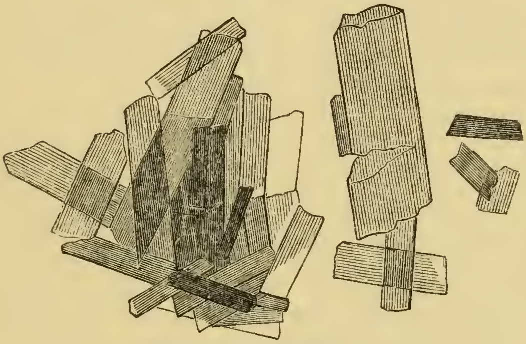
CAUSES OF HÆMORRHAGE.

Beside direct violence or injury, these causes can only be increased blood-pressure within the vessels or diminution in the power of resistance possessed by them; but a peculiar constitutional vice or diathesis exists in some persons which predisposes them to bleeding, of which the cause is not always obvious.

The fluid pressure exerted by blood within the vessels is of course increased both in active and passive hyperæmia, and hence either of these conditions may be the cause of hæmorrhage. As examples of the former, we may quote the hæmaturia produced by diuretics, and probably the common occurrence, bleeding from the nose; of the latter, hæmorrhage from the stomach in cases of stasis in the portal vein, or from the lung in cases of obstructive heart disease, are good examples. There are various alterations in the walls of vessels which diminish their power of resistance to the blood pressure; such are fatty degeneration and atheroma, causing rigidity of the walls, with or without dilatation and aneurism, which occasion hæmorrhage in the brain and in the heart. Similar alterations are, according to Cohnheim, the cause of the hæmorrhage which follows the obstruction of small arteries (*hæmorrhagic infarctus*). The vascular walls become altered by the failure of nutrition due to the absence of arterial blood. The vessels of new growths, as tumours and granulations, are often very delicate and liable to rupture, and the same is true of the vessels in new-born infants, whence arises their great liability to hæmorrhage. Inflammation of certain parts, as the lungs, is accompanied by hæmorrhage. The peculiar liability to hæmorrhage seen in certain individuals and in certain diseases, is not easily explained. Sometimes it seems almost to constitute a disease by itself, as purpura, or is combined with more serious cachexia as in scurvy; or gives a peculiar character to some cases of other diseases, as hæmorrhagic small-pox, hæmorrhagic scarlatina, &c. These phenomena have been supposed to depend upon some (chemical) change in the composition of the blood, and though this has not been demonstrated, certain experiments have thrown light upon it. Thus, poisoning with phosphorus or with sulphuric acid produces hæmorrhage, and the injection of solution of common salt into the veins of frogs has been seen to cause the passage of red blood corpuscles through the walls. Some persons again have the remarkable constitutional peculiarity, not only that they are particularly liable to hæmorrhage, but that bleeding, once set up in them, cannot be stopped. This curious tendency, which has received the name of "hæmophily," appears to be often hereditary, and to be especially derived from the female parent. It affects males much more than females, and more severely. Dr. Copland states that hæmorrhages are more common in the offspring of parents who have suffered from them than in others, and that the tendency is observed in several members of the same family. Hæmorrhage from the rectum, urinary organs, and uterus, is said by Chomel to occur oftener in cold than in warm seasons, and epistaxis and hæmoptysis to be more frequent in summer than in winter. Dr. Prout observed a peculiar tendency to renal hæmorrhage during the time that cholera was prevalent. Age has an influence in determining the seat of hæmorrhage. Epistaxis is most common in children, hæmoptysis in the early period of life, and hæmorrhoidal discharge and apoplexy in the aged.

Changes in the Effused Blood.—The blood, when effused, may remain in a liquid state for some time, or quickly coagulate. When it is poured out into the substance of a part, it undergoes, after a time, the changes which are commonly observed when a superficial part has been bruised. These consist in alteration of the colour of the hæmoglobin, which passes "from a dark-red into a blue, then into a brown, and lastly into a yellow colour, before it entirely disappears." At the same time the blood-globules, at least in many cases, undergo peculiar changes; they become massed together, and sometimes included in a kind of cellular envelope; they waste and shrink up, until there remain at last only minute yellow or orange-red granules, which evidently consist chiefly of pigment. Such are not unfrequently found in the straight tubuli of kidneys affected with morbus Brightii; they are the undoubted records of former hæmorrhage. Black pigment may also be the final stage in the transformation of hæmoglobin. In some cases the altered hæmatine takes the form of crystalloid, elongated, rectangular tablets, which vary very much in size, and are coloured more or less deeply by red matter. The formation of these seems to be promoted by the addition of water. They were extremely well seen in a case which

FIG. 4.



Hæmatine crystals.

we witnessed of cystic disease of the kidney, in which several large dark clots were contained in a cyst of extraordinary magnitude. The fibrine, and other residue of the extravasation, together with broken-up fragments of the tissue, are gradually reabsorbed, the solid substances undergoing liquefaction, chiefly in the way of fatty transformation.

Formation of Cysts.—A further change may take place, not so much in the effused blood, as in the parts around it. These, which are at first ragged and torn, undergo more or less inflammation, which ends in the effusion of a solidifying blastema; this fibrillates, and passes into the state of more or less perfect fibrous or areolar tissue, and thus forms a capsule or cyst, enclosing the now more or less altered blood. Rokitansky describes the cyst as being lined by a coloured, soft, gelatinous, loosely-adherent layer, formed from the coagulum, which, at a later period, by fibrillating, and even developing vessels, assumes very much the aspect of a delicate serous membrane. The contents of the cyst may be a gelatinous or serous fluid alone, or with more or less traces of a vascularized areolar tissue. In some cases absorption takes place completely, and the cavity is obliterated by the adhesion of the opposite sides, and the formation of a linear cicatrix. This, however, is not the most frequent issue, in consequence of the following impediments:—(1) a large size of the cyst; (2) retraction of the surrounding tissue, depending partly on its atrophy, partly on its induration; (3) the deposition of the fibrine, either as a central lumpy mass, or as a thickish, peripheral, capsulating layer.

Hœmatoma.—The effused blood-mass may undergo a different kind of change, in consequence of absorption of its watery parts, and become, in this way, a kind of tumour, termed an hæmatoma, classed with new growths; but there is no doubt that it is a simple result of hæmorrhage. and this for three reasons:—(1) that it presents no higher structural character than that of fibrine; (2) that it is generally devoid of vessels; (3) that it does not appear to increase by growth in the proper sense of the term. The blood remains in these masses uncoagulated for a longer or shorter time. The inner parts ultimately undergo some form of degeneration, while the outer form a fibrous investment. Hæmatomata occur in serous and synovial cavities, beneath fibrous and mucous membranes, in parenchymatous organs, in the muscular masses of the limbs, in the substance of certain new products, especially encephaloid cancers, and in cavities accidentally formed in the tissues, as in tuberculous cavities of the lungs. An hæmatoma thus formed being essentially a fibrinous mass, may undergo certain other changes; saline earthy matter may be deposited in it, inducing a state which is more correctly named *cretification*, than ossification; melanic pigment may probably also form in it. It may, perhaps, undergo a development to the somewhat higher stage of fibrous tumour, and it is said that even true bone may be formed within it. A vascular plexus has been observed in several instances in tumours of this kind; and though it may be objected that this has been developed in superadded exudation-matter, yet it appears to us very much more probable that the persistent fibrine afforded the developmental nidus from its own substance. Ultimately degeneration and liquefaction of the enclosed blood may take place; but these changes are very slow. Virchow found altered but recognizable blood-corpuscles in the contents of an hæmatoma formed three years and-a-half before death.* The most remarkable hæmatomata are probably those formed in the cerebral arachnoid.

Flux and Dropsy.—We have already in part noticed these results of hyperæmia, more particularly of the passive variety; but it seems desirable, on account of their great importance and frequency, to review them separately. The term flux may be properly applied to a discharge of various kinds taking place from a mucous surface, or from a glandular organ connected therewith; the term dropsy to an effusion of fluid in serous or synovial cavities, or in the areolar tissue. Fluxes will be active or passive, according to the kind of hyperæmia which occasions them; the same can scarcely be said of dropsies; the very great majority of them are passive. It is necessary to fix some limitation to the kind of fluid that may be said to constitute flux or dropsy, as there are many exudations which require to be distinguished on account of their different nature. A sero-purulent effusion, on the secretion of a serous cyst, would not come under the present head. Perhaps we shall be nearly correct if we say that a fluid similar to, but more aqueous than the liquor sanguinis, mingled in the case of flux, with a varying quantity of mucous secretion or desquamated epithelium, mature or immature; and if proceeding from a gland, mingled with more or less of its secretion, is that which properly belongs to this kind of morbid action.

Active Flux.—Of active fluxes we have a good example in miniature in a common sneeze; the morbid sensation which arises in the Schneiderian membrane causes at the same time inhibitory paralysis of arteries and capillaries, and contraction of the expiratory muscles. More considerable and much more enduring is the hyperæmia in the state of coryza, and the serous flux is of course much more prolonged. In this instance we have an opportunity of observing a quality of the fluid of a serous flux which is very common, viz., that it is especially acrid and irritating, so that it will sometimes excoriate the parts over which it flows. What gives it this quality is not very apparent; it seems at least doubtful whether it is merely an excess of the natural saline ingredients of the blood. We should rather suppose it to be some organic acid salt, of new formation. The fluid under the microscope exhibits very little trace of corpuscles, and is alkaline.

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* Virchow. "Die Krankhaften Geschwülste," Bd. I. s. 144. The whole chapter gives a good account of hæmatoma.

Choleraic diarrhœa is the extremest example of active morbid flux; the whole blood seems to rush to the intestinal surface, and pour out its fluid part, minus the greater part of the albumen and fibrine. The gruel-like evacuations consist of water and saline matter, with some trace of albumen in solution, and a large quantity of columnar epithelium. The reaction of the fluid is alkaline. The filtering action of the intestinal membrane in this instance is very marked, and well worth noticing; it is, indeed, extraordinary that, while so rapid a rush of blood is going on to the exhaling surface the effused fluid should be so considerably altered from that which arrives thither; one would have expected it to contain at least as much albumen as the passive exudation of ascites. We would ask whether the case of choleraic diarrhœa, as well as the similar condition from drastic purgatives, do not absolutely prove the existence of a power influencing the circulation other than the *vis à tergo* of the heart, with the regulating contractility of the blood-vessels? Does it not also demonstrate that it is this nutrition force of the tissues, as we have called it, which determines whether an hyperæmia shall issue in a flux or in an inflammation, in cholera or enteritis? In profuse salivation, arising from the administration of mercury, or other causes, we have a good instance of an active flux, from a glandular organ, although the fluid is in this case almost identical with the natural secretion. The characteristics generally of active fluxes are those of active hyperæmia, which is more or less apparent according to the amount of the effused fluid; if this be considerable, the hyperæmia is dissipated as fast as it arises.

Active Dropsy.—Active dropsies are often termed acute or febrile, and are not always easily distinguished from inflammatory effusions. An almost certain means of distinction is to observe whether the effused fluid is even slightly turbid with flakes of lymph, or puriform corpuscles; the presence of these is decisive of the inflammatory nature, or at least of some degree of co-existing inflammation. Acute anasarca affords one of the best instances of active dropsy, the interruption of the action of the kidneys, at the same time that it deteriorates the quality of the blood, and renders it less fit to circulate in the vessels, diminishes considerably the separation of fluid from it, so that from both these causes there arises a tendency to the effusion of fluid in the areolar tissue, or in other parts. The tenseness and firmness of the anasarcous swelling in many of these cases lead to the belief which direct observation has confirmed, that the effused fluid contains some amount of fibrine, which coagulates among the elementary parts of the tissue, and makes them more dense and stiff. The same thing occurs also in dropsies of serous cavities, and has been particularly noticed by Vogel, under the name of *Hydrops fibrinosus.* Most of these, however, are essentially inflammations, though of a mild type.

Coagulation of Serous Effusions.—It has been shown of late years that while the fluid of serous effusions is very rarely spontaneously coagulable, it nearly always contains one of the two constituents, by the combination of which fibrin is formed, namely, the *fibrinogenous* substance of Schmidt. This substance, when mixed with *fibrino-plastic* substance, or paraglobulin, as for instance with blood-corpuscles, immediately coagulates into fibrin. An exceedingly small quantity of paraglobulin is sufficient. Schmidt only failed to produce this result in eleven out of ninety-three cases; and in several of these exceptional cases it was evident that coagulation had already taken place within the body. It does not seem possible to draw any clear line between those exudations which do and those which do not spontaneously coagulate. A slight admixture of blood may of course produce the former result; and so may post-mortem transudation from the vessels.

Passive Flux.—Passive fluxes are of extreme frequency, and are almost invariably associated with debility. Their copiousness, the aqueous nature of the fluid, the frequent pallor, and non-elevated temperature of the parts from whence they proceed, as well as relaxed condition of these, may be said to be their general characters. They take place, as is manifest, from mucous surfaces, or from the glands that open upon them, and are, in consequence, mingled, more or less, with liquor muci, and with epithelial particles, in various stages of formation. In some cases, as in bronchorrhœa, occasionally they may depend upon venous obstruction; but their most essential cause seems to be a peculiarly relaxed and toneless state of the walls of the vessels, and of the tissues affected. This is confirmed by the beneficial effect of astringents, locally applied. At the same time, there is no doubt that the state of the blood, and of the system generally, has an influence upon them, and that as the quality (the crasis) of the former is improved, so the debilitating profluvium will diminish. It is reasonable to suppose that in this way the walls of the vessels are brought into a condition of more healthy tone. The peculiar condition of the tissues, as to their vital endowments, seems to be the only sufficient cause to which we can at all ascribe the different phenomena exhibited by the same tissues, under similar circumstances. One person shall have a chronic bronchitis, with puriform expectoration, while another person is suffering under bronchorrhœa, although the circumstances may be similar, and the two affections very much alike in their outset. When we speak of a toneless and relaxed condition of the vessels being the main cause of the flux, we do not so much mean to imply a defect in their contractile power (though this doubtless exists), but rather such an alteration of their texture, as that they are much more transudable by the aqueous part of the blood than is normally the case. In health a certain slight exudation takes place from the capillaries and all the minuter vessels, forming a nutrient atmosphere, in which the tissues are bathed. The exact composition of this is uncertain; but as the following experiment shows, it probably contains less albumen and fibrine than the liq. sanguinis. Valentin, having made an albuminous solution of a certain specific gravity, placed it on a filter of stretched serous membrane; and, on examining the fluid which passed through, he found the specific gravity reduced—that is to say, some of the albumen was left behind. It is probable, and seems proved by pathological experience, that increase of the pressure upon the fluid causes it to transude in a less altered state, so that this, whethor it be a *vis à tergo*, from increased cardiac impulse, or caused by an obstruction in the onward direction, must be one cause of an effusion containing much albumen and fibrine also. But the common watery and mucous effusions, which constitute such fluxes as those of leucorrhœa and bronchorrhœa, contain little, if any, albumen, and no fibrine, and are chiefly remarkable by the quantity of their aqueous and saline contents. The same may be said also of the intestinal and cutaneous fluxes that take place in phthisis, and in other exhausting diseases.* In all these it seems certain that the natural filtering power of the walls of the vessels is changed in such a way that they allow the aqueous and saline part of the blood to transude with extreme rapidity. At the same time the crasis of the blood itself is altered; it turns, as the popular phrase is, to water—*i.e.*, its corpuscles and its organic matters are not formed in due proportion; but, on the contrary, waste and diminish, so that the colliquative discharges from the tissues are promoted, and kept up by the (as it were) deliquescing blood. It is interesting to observe that the filtrating property of the vascular membrane is capable of being influenced through the nervous system. Thus, after a fit of hysteria, a quantity of limpid aqueous urine is passed, much more than would have been voided had no such event occurred. This must depend upon an alteration of the condition of the Malpighian tufts, and perhaps of the capillaries of the tubular venous plexus. We have noticed something of the same kind after a small dose of opium. Almost the only instances of fluxes taking place from glands, with which we are acquainted, are those which the kidneys afford. That of diabetes depends, as is well known, upon a diuretic substance, sugar circulating in the blood, and not undergoing the decomposition which it should normally. Discharges, such as those of chronic bronchitis, or chronic dysentery, which were, in their commencement, of truly inflammatory nature, but afterwards become more of the nature of fluxes, often contain a very large amount of muco-purulent matter, and cause a proportionally severe drain on the system. These, however, do not present the hydræmic condition of the general system, before alluded to.

Passive Dropsy.—Passive dropsies are the commonest of all; all the cardiac dropsies, and most of the renal that we meet with are of this kind. Their very aspect excludes the idea of increased action (however the term may be understood), and naturally suggests that of some obstruction to the circulation, with diminution of the vital energies. The surface is generally pallid, or of a dull venous hue, the animal heat is diminished, the anasarcous swellings pit easily on pressure, the effect of gravity upon them is marked, the movements are languid, the respiration often embarrassed, and the mind depressed. One of the most common, but as we shall see, probably, not their immediate cause, at least in many instances, is, beyond doubt, a mechanical impediment to the free course of the blood. This was long ago proved by the well-known experiment of Lower. He tied the jugular vein of a dog, and found that all the tissues of the head and face were infiltrated after the lapse of some hours, not with extravasated blood, as he had rather expected, but with clear serum. The analogous instance of ascites resulting from cirrhosis of the liver has been already mentioned. Œdema has also, beyond doubt, a very close connection with the lymphatic system; and some have gone so far as to speak of it as a lymphatic congestion. Ludwig saw reason to believe that an artificially produced œdema of the lip of a dog was completely emptied by the lymphatic channels. These phenomena doubtless depend upon the continuity of the spaces of areolar tissue with minute or capillary lymphatics. The same explanation applies to accumulation of fluid in serous cavities, which have been recently shown to stand in open communication with the lymphatics; and this may also depend upon obstructed or imperfect removal of fluid by the lymphatics. It does not follow that in either of these cases the fluid accumulated is precisely normal lymph, since it is more than probable that it undergoes pathological modifications.

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* Simon states that he failed in detecting any certain indications of albumen in the sweat collected (by means of linen washed with distilled water) from the breast of a person in the colliquative stage of tubercular phthisis.

Conditions of the Occurrence of Dropsy.—But it requires no very long pathological experience to discover that there are cases not unfrequently occurring in which, although there exist abundant causes of obstruction to the circulation, yet dropsy does not take place. Dr. Walshe, in his work on Diseases of the Lungs and Heart (page 649, 2nd ed.), has given a list of most serious diseased conditions of the heart which may exist without producing dropsy, and concludes justly, that something beyond all these is wanting to insure this occurrence. What, then, is this? Andral shows, in his "Hématologie," that neither diminution of the globules, nor of the fibrine of the blood, is the immediate cause of dropsy, but that this always accompanies a diminution of the albumen. It is therefore highly probable that it is this alteration of the crasis of the blood which determines the occurrence of dropsy in persons who are predisposed to it by organic disease of the heart or lungs, which causes congestion of the venous system. It is not only in dropsies of cardiac origin that diminution of the albumen seems to be the most important moment* in producing the effusion. This cause is evidently influential in renal dropsy, in which a constant drain of albuminous serum out of the blood is taking place. It is remarkable that this may have been going on for some, perhaps a considerable time, and yet no dropsy occur. The explanation of this is afforded by the circumstance ascertained by Simon and Christison, that the decrease of the solid constituents of the serum is not always the leading character in this disease. In three of Simon's analyses out of four of blood in Bright's disease, the quantity of albumen was decidedly increased —in one instance amounting to 109·4, considerably above the average of health. Cases of dropsy occasionally are met with, in which, as there appears no absolute organic disease, but only an hydræmic condition of the blood, one is obliged to conclude that the effusion is dependent on this. Andral mentions that during a famine, where the poorer classes had been obliged to seek a scanty nourishment in roots and herbs growing in the fields, many persons became dropsical. This has not always been observed in famines; but a curious observation of Brücke's bears upon this point. He confined a frog, having the sciatic nerve of one leg divided, in a glass vessel for some months. When the frog was insufficiently fed œdema occurred in the leg of which the nerve was divided; abundant animal food removed the œdema, which reappeared when the animal was again starved. The occurrence of œdema, in the affected leg only, illustrates the effect of the abolition of nervous influence in causing local dropsy; the same thing is sometimes, though rarely, observed in infantile paralysis. Dr. Laycock has repeatedly drawn attention to similar facts.

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* We ask leave to introduce this word as a convenient term, signifying an influential condition.

In these instances, it is very probable that the proportion of albumen in the blood was diminished, as it is clear that the supply of it ordinarily derived from the food was so. When, from cardiac or renal causes, or both combined, together with altered crasis of the blood, the tendency to dropsical effusion is very strong, it is quite remarkable how universal the dropsy becomes, the peritoneum, both pleuræ, and the pericardium, may be found full of fluid, the areolar texture everywhere infiltrated, the air-cells of the lungs loaded with frothy serum, the tissue of the brain "wet," and the subarachnoid fluid considerably increased. In fact, it seems as if the vessels no longer presented any containing barrier, but permitted the escape of fluid in every part that it traversed. It is often observable in these cases, after death, how the naturally transparent serous membranes have lost this appearance, they look thickened, of a dull whity grey tint, as it were sodden in the fluid. There can be no doubt that this depends on a chronic thickening and increase of their fibrous layer.

Composition of Dropsical Effusions.—This generally approaches more or less closely to that of the serum of the blood. The purer fluids are clear, tolerably limpid, and colourless; often, however, a marked yellow tint is observed, which may either arise from dissolved hæmatine or from the presence of an increased quantity of the natural yellow pigment of the serum; or again, especially in the case of ascites, from dissolved bile pigment. If the latter is the case it will be rendered evident by the reaction with nitric acid. A milk-white turbidity is occasionally observed, which depends on the admixture of fat (oil) or epithelium scales; or, according to Schmidt, of a peculiar albuminous substance. Blood globules may be often seen in great numbers in the fluids of ascites by the aid of the microscope; lymph corpuscles or leucocytes are always present; cholesterine tablets are common in that of hydrocele, and may be sufficiently numerous to constitute a crystalline deposit. The reaction of the fluid is alkaline; in rare instances an acid has been observed. Lactic, hippuric, and uric acids have been found. Urea is always present, usually in minute quantity, but as much as 6 per cent. has been determined. Sugar is found in cases of diabetes. The salts are principally chloride of sodium and the other salts of the blood serum, nearly in the same proportions as in that fluid. The gases of the blood (oxygen, nitrogen, and carbonic acid) have also been found in minute quantities. The proportion of water varies a good deal, though almost always greater than in the blood serum, while the amount of albumen shows converse variations. We subjoin from Vogel the results of seven analyses, which show some remarkable variations in the amount of the several constituents.

	1	2	3	4	5	6	7
Water	905·0	920·0	927	946	956	988·0	704
Albumen	78·0	71·5	48	33	29	0·9	290
Extractive matter	4·2		10	13	9		2
Fat	3·8		9		7	10 0	
Salts	9·0	8.5	6	8	8		4
	Blood Serum.	Hydrocele	Hydrocele	Ascites	Ascites	Ascites	Ascites

The seventh analysis shows actually a larger amount of albumen than is present in the serum of the blood; this might be supposed to be an error, if other similar instances had not been observed; it probably depends upon a quantity of the water of the original effusion having been removed by absorption, so that the fluid became more concentrated. The most recent determinations, given by O. Weber,* do not differ from these in any important respect.

PNEUMATOSIS.

The occurrence of air or gas in various parts of the body was at one time thought to be a much commoner event than it is now admitted to be, and its production was spoken of as a well-recognized morbid process, under the name of Pneumatosis. Lately much doubt has been thrown on the very existence of such a process; and the abnormal collections of air are traced to one of the natural air-containing cavities, the lungs and air passages, or to the stomach and intestines, which may contain air swallowed with the food, or gases arising from its decomposition. Many cases, such as the presence of air in the meninges, the pericardium, the mediastinum, are known to be explicable by the entrance of air during the dissection. Air in the pleura, it is generally admitted, can come only from the lungs; in the peritoneum (for the immense majority of cases) from the intestine, only in the rarest instances gas being actually liberated there by the decomposition of sanious pus, or similar matter. We have a right, then, to regard with the greatest suspicion all cases of apparently spontaneous production of gas, and to exhaust, as far as possible, other explanations before resorting to the hypothesis of its actual generation.

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* Pitha and Billroth, "Handbuch der Chirurgie," vol. i.

It must be admitted, however, that it is not always easy to explain the origin of gaseous accumulations, and the obscurity of such cases gives them an interest out of proportion to their pathological importance. In the Hunterian Museum are preserved some classical specimens of gaseous cysts, of which it has been said that they "should be admired, or rather venerated, for their histories include the honourable names of Hunter, of Jenner, of Cavendish" (Sir J. Paget). These cysts were attached to the intestine of a hog, and were sent by Jenner to Hunter. Cavendish analysed the air contained, and found what in the language of modern chemistry would mean atmospheric air with excess of carbonic acid and diminished proportion of oxygen. No explanation is given of their occurrence, but they were said by Jenner to be common in the intestines of hogs killed in the summer months. The editor of this work met with a very similar specimen in the peritoneum of a man who died with ulceration in the stomach, where small cysts containing gas were attached to the outside of the intestines. The gas was found, as in the case just mentioned, to consist of atmospheric air with excess of carbonic acid and marked diminution of oxygen. In this case it was thought that air contained in the stomach (which was greatly distended from pyloric constriction) had found its way, by means of the ulcer, between the coats and along the intestinal wall, till it dilated the serous covering into the form of cysts; the latter becoming in the end permanent structures. In another case, a similar subperitoneal emphysema was observed as a consequence of cancer of the œsophagus, and probably from a similar cause.* It is of course always important to determine the absolute impossibility of the gas arising from post-mortem changes, since an appearance called post-mortem emphysema is not uncommon: where gas is produced in numerous small cavities in the viscera by rapid putrefaction, aided by bacteria. This occurs especially in gangrenous or septicæmic conditions, and of course more often in hot weather.

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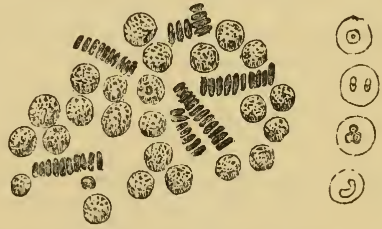
* Payne: Trans. Path. Soc., xxii. 336. 1871.

LEUCOCYTHÆMIA OR LEUCHÆMIA

are names given to a condition of the blood in which the white corpuscles are enormously increased in number. The former name is the more correct, as the blood does not lose its red colour, and as the prominent alteration in it is the great increase of white corpuscles. When a drop of blood, drawn during life, is examined microscopically, the red corpuscles appear tolerably natural, and often arranged in rouleaux, leaving intermediate spaces, which are more or less crowded with the white corpuscles. It is difficult to say what is the proportion which the one set of corpuscles bear to the other. In cases which we have witnessed, we should have regarded them as nearly equal, comparing mass to mass, or thus estimated, the colourless may, from their larger size, preponderate. Dr. Bennet estimates the white as scarcely one-third the number of the red, which seems about the average proportion in advanced cases. Many of the white corpuscles are very much larger than the natural size. They have more coarsely granular contents than the normal ones, with an interior single, double, or tripartite nucleus. The envelope and nucleus are brought into view distinctly by the action of acetic acid, which renders the granular contents transparent. Occasionally a crescentic nucleus is to be seen in the cells, and some free nuclei also are observed between them. In rare cases, coloured nucleated corpuscles, or transitional forms between the white and the red, have been observed. The blood, in fatal cases, is often found imperfectly coagulated—sometimes grumous, of a dirty brown colour. The coagula, where decolorized, have not the aspect of healthy fibrine, but are of a more opaque dull yellow, and, when broken up, resemble thick creamy pus. They contain in this part very numerous white corpuscles, to which the peculiar aspect is no doubt due. The red corpuscles are invariably diminished; the solids of the serum little altered; but the specific gravity of the blood shows a constant diminution (from 1055 to 1040 or 1035). The iron is also deficient from the paucity of red corpuscles. In some cases the blood has had an acid reaction, and has contained xanthin and hypoxanthin (bodies also obtained from the spleen), together with gelatin and some other nitrogenous substances of simpler constitution, and lactic acid.

Fig. 5.



Blood in Leucocythæmia—four of the white corpuscles have been treated with acetic acid. From Dr. H. Bennet's work.

Morbid Anatomy.—Morbid changes are chiefly observed, in the first place, in the spleen and the lymphatic glands; secondly, in various organs which are regarded as resembling lymph-glands in structure, viz., the tonsils, and other follicular structures of the intestinal tract, and the red medullary tissue of bones; and finally, in very different organs, as the liver, kidneys, lungs, &c. Some of these morbid changes are regarded as *primary*, being thought to be the cause of the state of the blood; others are called *secondary*, being regarded as its consequences.

Primary Morbid Changes.—The researches of numerous pathologists, more especially of Virchow,[*] Bennet,[†] Neumann,[‡] Mosler,[§] and Béhier, have shown the very close connection of this disease with changes in certain organs, which are believed to take part in the formation of the colourless corpuscles of the blood, viz., the spleen, the lymphatic glands, and the red medulla of bones, as well as, in an inferior degree, other lymphatic structures, such as the follicles of the digestive tract. The superabundance of white corpuscles in the blood appears to be a consequence of hypertrophy of these structures, which in such a condition furnish to the blood a much larger number of corpuscles than they do normally. There may, however, be hypertrophy of these structures without leucocythæmia, and the precise nature of the difference between these classes of cases is not clearly understood.

According to the primary morbid change which is the starting-point of the disease, Virchow has distinguished two forms of leuchæmia, viz., the splenic and the lymphatic; in the one the spleen, in the other the lymphatic glands, being the starting-point; but cases in which either of these organs is exclusively affected are uncommon, most being what Virchow calls mixed cases. In purely lymphatic leucocythæmia the colourless elements of the blood are said to be somewhat smaller than ordinary white corpuscles, but to possess larger nuclei; while in the purely splenic cases they resemble the normal corpuscles of the blood, except in being for the most part larger, and usually containing multiple nuclei. The so-called lymphatic form is, however, extremely rare. To these two types must now be added, for the sake of completeness, two others, viz., the form which arises from an affection of the osseous medulla, first observed by Neumann; and another, in which the blood changes are dependent upon hypertrophy of the glandular structures of the intestine, seen as yet in one case only, by Béhier. The first of these forms has been called the *myelogenous* by Neumann. He was the first to show that the red medulla of bones has many characters of a lymphatic organ, and is in all probability one of the parts where both red and white blood-corpuscles are formed. In several cases of leucocythæmia changes have been found in this tissue showing an increased production of white corpuscles; and though in most of these there were also characteristic changes in the spleen and lymphatic glands, one remarkable case has been recorded in which, together with some swelling of the spleen, there was immense enlargement and hyperplasia of the medulla of several bones, so that the latter change could not but be regarded as the predominant one.*

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[*] "Gesammelte Abhandlungen," 1856; "Die Krankhaften Geschwülste," vol. ii.; "Cellular Pathology," translated by Chance.

[†] On "Leucocythæmia," 1852.

[‡] "Archiv der Heilkunde," 1869, vol. x. p. 68, and 1870, xi. p. 1. See also "Quarterly Journal of Microscopical Science," 1870: art., "Neumann's Researches," and 1871, p. 292.

[§] "Die Pathologie und Therapie der Leukämie," Berlin, 1872. The standard work on the subject, containing very numerous references.

The last mode of origin of leucocythæmia is from the follicular (lymphatic) structures of the intestines. Although these parts are often the seat of secondary morbid changes, only one case has been observed in which they appeared to be the starting-point of the disease. This observation is due to M. Béhier.† In a man, aged twenty-four, of extremely anæmic appearance, there was found decided leucocythæmia, the white corpuscles being almost as numerous as the red. These corpuscles were of moderate size, as in the lymphatic form of the disease, and smaller than those found in the splenic form. After death no enlargement of the spleen or lymphatic glands, nor any notable change in any organ, except the Peyer's patches and solitary glands of the small intestine, which were much swollen and enlarged by an over-growth of lymphoid tissue, though not ulcerated. No other part being diseased, it was concluded that the excess of white corpuscles was derived from the intestinal follicles; but it is true that the bones were not examined.

Histology of Primary Morbid Changes.—The enlargement of the spleen is found to be due to hypertrophy of the proper spleen pulp, the Malpighian follicles being comparatively unaffected, or even small. In early stages the cells of the pulp are chiefly increased; but later on there is also induration from hypertrophy of the fibrous elements. At the same time exudation takes place, and masses are formed resembling blocks, or infarctions, which may undergo degeneration. In the lymphatic glands the enlargement of the cortical portion, and especially the multiplication of its cellular elements, predominates.

Secondary Morbid Changes.—The secondary changes consist chiefly in the formation of tumours, or growths, of a lymphatic character (lymphomata), consisting chiefly of lymph-cells, with a more or less clearly developed stroma or reticulum. Beside actual tumours, diffuse infiltrations of the same kind are met with, and every transitional form between the two may be found. From a study of the latter, it is clear that the morbid change begins in all organs in the interstitial connective tissue, or stroma, not in the special secretive or glandular elements. Thus, in the kidney white corpuscles are found between the tubes, and around the Malpighian bodies. The origin of these infiltrations is not always clear, some authorities regarding them as produced by emigration of the leucocytes from the blood-vessels; others, as derived from proliferation of the connective tissue corpuscles. The new growths bear a close resemblance to the structure of a lymphatic gland, and are perhaps due to the enlargement of previously existing adenoid tissue. It is probable that many of the so-called secondary growths are really primary, and contribute, like the enlarged spleen and lymphatic glands, to the excess of leucocytes in the blood.

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* Waldeyer. Virchow's "Archiv," vol. lii. 3.

† "A Contribution to the History of Leucæmia." Paris, 1868.

The secondary changes are found most commonly on the mucous membrane of the intestines; sometimes on the respiratory mucous membrane; and, further, in the liver and kidneys. More rarely they have been found in the heart, the retina, the suprarenal capsules; also in the thymus, thyroid, follicles of the tongue, tonsils, and, doubtfully, in the lungs. The organs in which these changes are found are usually abnormal in other respects also. The liver is often very greatly enlarged, and this not so much by lymphatic formations as by enlargement and multiplication of the liver cells. In the kidneys (which are also often enlarged) we find swelling of the epithelium in the uriniferous tubes; sometimes proliferation, and other marks of inflammation. In mucous surfaces the swellings first produced by enlargement of the follicular structures may soften, and lead to ulceration. In the retina, beside minute lymphatic formations, are seen remarkable paleness of the vessels, and peculiar white spots. These changes, constituting the *Retinitis leukœmica* of Liebreich, may be observed with the ophthalmoscope.

Symptoms and Causes.—The coming on of the disease is usually very insidious. Emaciation, weakness, extreme pallor, and other signs of anæmia, are early symptoms. Dyspnœa is also frequently noted, and later on hæmorrhage from various parts (especially from the nose), and dropsy. In women amenorrhœa, and other disturbances of the sexual functions, are observed. Diarrhœa is a frequent symptom; vomiting is less often present. The affection has been more often observed in males than females, in the ratio of 16 to 9. It seems "to be most common in adult life, and more frequent in advanced age than in youth." Some febrile disturbance is not unfrequent, but not to any great degree, or of long continuance.

Little is known of the causes of this disease. In a few cases there has been an antecedent history of syphilis; in others, of ague (though the ordinary enlargement of the spleen in the latter disease must not be confounded with the leuchæmic enlargement); and, occasionally in women, some disturbance of the sexual functions, such as exhaustion after parturition, or repeated miscarriages. But in the great majority of cases none of these influences have been traced, and the malady has remained quite unexplained.

The prognosis is extremely bad, no case of recovery being known when the disease was well established.

CHAPTER III.

INFLAMMATION.

Familiar as are the phenomena, and time-honoured as is the name of inflammation, authorities are not precisely agreed as to its definition. Formerly the conception was entirely derived from superficial characters; lately endeavours have been made to give it precision, by selecting, and perhaps exaggerating, some of its phenomena. Thus, Dr. Williams speaks of inflammation as a form of hyperæmia, in which motion of blood in the part is partly increased, partly diminished. Mr. Simon thinks that the entire process consists essentially in *local change of material*, involving simultaneous loss of substance and reproduction of substance; and differs from ordinary nutrition or life (of which this is also true), in being so rapid and intense, that the opposed results are *appreciable*. A more comprehensive definition is aimed at by adding to the conception change of nutrition that of *functional disturbance;* hyperæmia and exudation being regarded as usual but not essential accompaniments (Uhle and Wagner). More lately the notion of inflammation has been derived from its most frequent cause. In the words of Cornil and Ranvier, it is "the series of phenomena observed in tissues or organs analogous to those produced in the same parts artificially by the action of a chemical or physical irritant." The definition of Dr. Burdon Sanderson is to the same effect. "By the 'process of inflammation' I understand the succession of changes which occurs in a living tissue when it is injured, provided that the injury is not of such a degree as at once to destroy its structure and vitality." As to their cause, he divides inflammations into those of extrinsic and those of intrinsic origin; the former being those produced by direct irritation; the latter, those commonly called idiopathic, but more correctly *infective*, the cause being internal. The effects of injurious irritation, according to the same authority, are three: (1) Disorder of circulation; (2) transudation of the constituents of the blood; (3) altered mode of growth of the elements of the inflamed texture. Of these the second only is absolutely essential, the alterations of circulation only assisting, but by no means causing, exudation; while the changes in the tissues are the consequence merely of the exudation of blood constituents.

Finally, we may sum up the phenomena which every complete definition of inflammation has to account for thus, as, (1) Active hyperæmia, or determination of blood; (2) Stagnation of blood; (3) Escape of the liquid and corpuscular constituents of the blood; (4) Changes in the tissues both in the direction of overgrowth and in the direction of degeneration or loss of substance; (5) Nervous disturbance, as pain and hyperæsthesia.

External Characters.—The general appearance of an inflamed part is well described in the terms handed down from the age of Celsus, as being the seat of redness, heat, pain, and swelling. These are the visible symptoms of a pathological process, which, though continually before our eyes, and of the utmost importance in its results, and though it has been the subject of numberless speculations and careful labours, we are compelled to acknowledge we are still imperfectly acquainted with. The redness of an inflamed part is more or less vivid; it is deepest in the centre, and gradually shades off towards the circumference. In this respect it differs from an extravasation, whose margin is more defined, as also in the circumstance that it can be in a greater degree removed by pressure, though by no means completely. The aspect of the redness may differ according to various circumstances; if the capillary networks of the part affected be plane, or uniformly extended, the injection will appear as an uniform deep blush; if, on the other hand, they are moulded to the form of villi, or folds of mucous membrane, the surface will have the appearance of a pile of red velvet: in fibrous structures a streaky appearance is observed, and generally the form of the redness will depend upon the arrangement of the capillaries of the part. The increased depth of colour is owing chiefly to distension of existing vessels, not in anywise to the formation of new ones, a process which does not take place till a much later period: the only other cause which at present exists is the staining of the surrounding tissues with exuded hæmoglobin, which may occur soon after stasis has been established. It is necessary to distinguish carefully between genuine inflammatory redness, and that which often simulates it closely in the dead body, viz., hypostatic, or depending solely on mechanical causes, or on the mode in which death has taken place. It is always desirable to take other circumstances into consideration at the same time, but we may generally say that we should doubt the inflammatory nature of a redness which existed solely in depending parts, or in those the large veins of which were much gorged, or which coincided with a fluid condition of the blood, and which was not attended with any thickening of the part. It may also be observed, that after an internal part has been exposed to the air a short time, it assumes a much more marked and brighter redness, which depends solely on the action of the oxygen in the air.

Temperature.—The natural temperature of an inflamed part seems to be considerably increased, and this as well to the sensations of the observer as of the sufferer (hence the name "inflammatio," a burning). The increase of heat is not always very great, as shown by the thermometer; in some of Hunter's experiments the difference was not more than one degree, and it seems doubtful whether the heat of the inflamed part is generally greater than that of the internal parts of the body. The temperature of an inflamed limb is always above that of a corresponding (uninflamed) part; but this difference has not been observed in an inflamed pleura.

The rapid interchange of blood must, of course, tend to equalize the temperatures of different parts of the body; but the later experiments of Simon* and O. Weber† have shown that the temperature in a wounded limb may be higher than that of the rectum, though from Jacobson's experiments this is not always so.‡ Simon's experiments with the thermo-electric apparatus, confirmed by those of Weber, have also shown that the arterial blood supplied to an inflamed part is cooler than the seat of inflammation itself; while the venous blood returning from the part, though warmer than the arterial blood, does not come up to the temperature of the inflamed part. Both the inflamed tissues themselves and the venous blood going from them were warmer than the corresponding parts of the other (normal) side of the body. There is always a general increase in the temperature of the body, as well as this local elevation; and from the results just quoted it was concluded that the general elevation was owing to the increased supply of heat from the inflamed part through the blood. Calculations, however, have proved that this explanation is not sufficient, and that local inflammation raises the general temperature in some other way, probably by producing the little-understood condition of general fever. With regard to the cause of increased temperature of inflamed parts, there can be no doubt that is due, in general terms, to the more rapid chemical change which accompanies, or constitutes, increased physiological activity; but no more definite explanation can, as yet, be given.

Pain and Swelling.—The pain of inflammation varies much in degree and in kind, according to its seat and intensity and exciting cause. That of inflamed serous membranes is often of a peculiar, sharp, darting kind; that of mucous membranes more dull and gravative, as it is termed; that of dense, unyielding, fibrous or bony textures, amounts sometimes to extreme agony. The inflammation heightens the sensibility of the nerves, which are at the same time compressed by the swollen textures and distended vessels.

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* Holmes's "System of Surgery:" article "Inflammation."

† "Deutsche Klinik," 1864, No. 43.

‡ Jacobson, Virchow's "Archiv," vol. li. p. 275. The results obtained by this experimenter, as well as those of Bernhardt and others, are at present in direct contradiction with those of Simon and Weber. See Virchow's "Jahresbericht," f 1869, vol. i. p. 124, f. 1870, vol. i. p. 188. "Journal of Anatomy and Physiology," May, 1871, p. 410.

Swelling depends manifestly in great measure upon the distension of the vessels with blood; the bulk of the part is increased just as that of a kidney or liver is when it is injected artificially—its fibres are put on the stretch, its vessels strained, and its capsule, if it have one, fully distended. The effusion of plasma is another cause of the swelling, especially at a later period and in some cases, but is not, we think, so powerful a cause as the vascular injection. A third cause of swelling is the production of new elements.

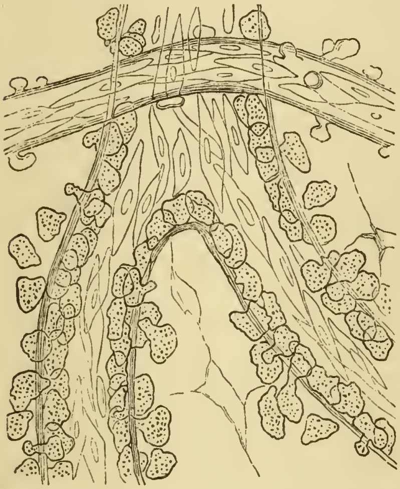
Vascular Phenomena. Stasis.—We now proceed to the more minute examination of the phenomena of the inflammatory process as they have been disclosed to us by the microscope, and we here resume the line of inquiry which we commenced during our consideration of active hyperæmia. We saw in this, that, with a certain amount of stimulation, the arteries enlarged, and admitted a greater quantity of blood, which flowed on more rapidly, and traversed with an accelerated current the capillaries and veins which became dilated also. An increased and more rapid blood-flow were then the characters of determination of blood. But if the stimulus is increased, or if it be excessive from the first, phenomena of a very different kind present themselves. The current slackens, it moves slower and slower, and at last ceases; the capillaries are seen distended with a red uniform mass, the veins are also enlarged, and filled with red corpuscles, crowded together, which retain more of their distinct form than those in the capillaries, and move either slowly onwards, or oscillate, or are quite stagnant; the arteries, which are also distended, exhibit for some time a progressive movement onward of their contents, which at first is steady, afterwards becomes jerky, or intermittent, and at last ceases. The condition of *stasis*, as it is called (ἵστημι, to stand), is now established, and therewith, as the sequence of active hyperæmia, inflammation. In the immediate neighbourhood of the seat of stagnation the circulation is still seen going on rapidly, and not only in the parts adjoining on the margins of the stasis, but even within its area capillary streams may be seen here and there rapidly coursing beneath a plexus of channels, which are filled by an uniformly red quiescent mass. Manifestly, determination of blood prevails actively all round the focus of arrest of movement. The stagnant blood in most of the capillaries presents an uniform red mass, in which the separate corpuscles are undistinguishable; here and there gaps may be seen, as if a fissure had taken place, and separated the adjacent portions a little away from each other. Amid the mass of blood-globules, appearing as it were fused together, may be seen occasionally one or two white corpuscles: according to our observation, they are certainly not to be seen in the great majority of capillary vessels. After the stasis has existed some time, they may be seen in great numbers coating the walls of the veins, and rolled along by the current passing through them; and occasionally they constitute, together with transparent plasma, the entire contents of a portion of vessel of some length, not remaining absolutely stagnant, but oscillating to and fro, or moving sometimes slowly onwards. From a vessel thus filled we have observed them escaping into a communicating vein, three or four at a time, and carried away into the general circulation. From repeated observations, we are quite satisfied that stasis is not produced in a mechanical sort of way by the corpuscles becoming sticky and adhering to the vascular walls and to each other. When weak liq. ammon. is applied to the web, and the actual occurrence of stasis watched, it appears as if the blood stream were suddenly arrested by some invisible power; the corpuscles, just before passing on so briskly, are retarded in their progress, more and more slowly, oscillate a little, then stop, accumulate, and become congested together into a mass. The adhesion of the corpuscles to the capillary walls is most marked after a short time; but it certainly is not the prime cause of stasis. On what the adhesion depends it is difficult to say; it has seemed to us more like the result of an attractive force than of mere stickiness. The diameter of the distended capillaries, which are the seat of stasis, has appeared to us, for the most part, tolerably uniform; but in one instance we observed numerous constrictions at various points. These were remarkably abrupt, and extended across one-third, or one-half, the channel. Mr. Wharton Jones mentions a local *dilatation* of arteries, but we find no account of these local contractions of the capillaries. The red globules, for the most part, appear to be packed together without any regular arrangement, but occasionally they may be seen lying together in rouleaux, like the corpuscles of human blood, with their long diameter transverse to the axis of the vessel. The white corpuscles are not unfrequently seen of a pyriform shape, dragging slowly along, or actually sticking to the sides of the vessels; that they do possess some degree of adhesiveness is manifest, but it does not seem to be so considerable or general as Dr. Williams supposes. When the inflammation is subsiding, and the stagnant blood beginning again to resume its course, all that can be observed is, that the agglomerated mass of red corpuscles in a vessel loosens and breaks up, so that the individual corpuscles are again visible, while the impulse of the heart makes itself more and more felt, and at last sweeps away the accumulation altogether, having first detached small portions successively. Fibrinous coagula also form occasionally, as Mr. Wharton Jones describes, and are similarly disintegrated, and carried away by the returning current during resolution of the inflammation. While stagnation continues, a small quantity of hæmoglobin dissolved in the serum exudes, and imparts to the tissues bordering the vessels some degree of yellow staining.

Later Observations.—We intend the foregoing account to serve as a description of what may be actually observed of the process of inflammation, as it occurs in the frog's web. Most of the statements we have verified by our own examination, and we believe they agree, so far as they go, with those of the best observers; but certain additional features have been described by later observers, who have chosen the mesentery, rather than the foot of the frog, to study these phenomena, and have used various improved methods. The following account is mainly that of Cohnheim,* but owes much also to the admirable summary of Dr. Burdon Sanderson.†

When the mesentery of a frog (the animal being kept at rest by poisoning with curare) is exposed under the microscope, the external air acts as a stimulus, and produces inflammation. The first thing seen is a dilatation of the arteries, which reaches a climax in about ten or twelve hours, when they may be double their original diameter; there is also a simultaneous elongation. A similar dilatation of the veins, but without elongation, follows after an interval. The velocity of the current is increased for a short time, but then becomes constant.

Thus far the phenomena are identical with those of active hyperæmia. But in inflammation the following changes are observed:—The current becomes slower (sometimes suddenly), loses its normal central or axial character, the corpuscles moving along in the whole breadth of the vessels, both arteries and veins. In the latter, however, the white corpuscles show a peculiar tendency to loiter, or hug the walls, till the inner surface of the veins becomes lined with rows of stationary white corpuscles, between which the current of blood continues. Next occurs the extraordinary phenomenon which Cohnheim has re-discovered and brought into prominence. The white corpuscles begin to penetrate the walls of the vein; small bud-like projections are seen on the outer side, which gradually enlarge while still connected with the wall, till at length they detach themselves and become free leucocytes. Others, meanwhile, leave the venous current, become attached to the wall, and penetrate it in the same way. The time necessary to set up this process varies much; sometimes it does not begin till

Fig. 6.



Migration of white and red corpuscles from a vein and capillary of the tadpole's tail. (Caton, "Journal of Anatomy and Physiology," vol. v., pl. iv., 1871.) The wall of the (branched) vein is seen to be penetrated by white corpuscles, while the red still move in the central stream. On the outside of the capillary (above) red corpuscles are seen projecting.

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* Virchow's "Archiv," 1867, vol. xl., p. 1.

† Holmes's "System of Surgery," vol. v.: article "Inflammation," p. 729, &c.

after many hours. Simultaneously with the veins, the capillaries also enlarge, but not more than one-sixth or one-fourth of their diameter ; in some the current goes on unchanged, while in others retardation of the current and emigration of the leucocytes take place as in the veins. Some red corpuscles also leave the veins and capillaries at the same time as the white, but not in the same numbers; they pass through slowly, and are sometimes broken in two by the blood current. There is also simultaneous exudation of blood serum. After a time (perhaps twenty-four hours) the capillaries and veins are closely surrounded with the emigrated blood corpuscles, and though these rapidly pass away, their places are taken by others.

The leucocytes, travelling partly by their own activity and partly perhaps conveyed by the exuded serum, at length fill the mesenteric tissue, and form layers on its upper surface, which may be solidified by the coagulation of the exuded serum.

Subsequent to this process, but in reality a part of it, is the phenomenon known as stasis, already described, which has its seat in the capillaries, and which therefore brings us back to the description formerly given.*

Causes of the Vascular Phenomena of Inflammation.—If we adopt the division of Dr. Burdon Sanderson (formerly given), we must consider the phenomena of inflammation under three heads—(1) Disorders of circulation ; (2) transudation of serum, emigration of cells, and stasis; (3) changes in the elements of the tissues. The causes of disordered circulation have already been considered under the head of Hyperæmia ; but those which come under the second head are peculiar to inflammation. These cannot be simply a consequence of the first class of phenomena, as then every active hyperæmia would result in inflammation : some other explanation is necessary, and the following have been suggested.

The view of Dr. Williams is that stasis and inflammation depend upon the white blood corpuscles. He maintains "that an essential part of inflammation is the production of numerous white globules in the inflamed vessels; and that the obstruction of these vessels is mainly due to the adhesive properties of these globules." With regard to this doctrine, it must be admitted that Dr. Williams anticipated some recent discoveries, in drawing attention to the behaviour of the white corpuscles; but neither the increased number nor the adhesiveness of these elements can completely explain the phenomena which have been described.

Other explanations formerly given need hardly be noticed now, since they are obviously untenable in face of the new facts which have come to light. There are, in fact, two distinct phenomena which have now to be accounted for—the emigration of cells, and the subsequent stasis.

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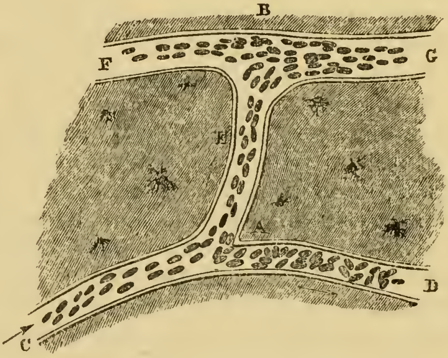
* It should be stated that emigration of leucocytes from the vessels appears to be a normal process in frogs, and also in some invertebrata; and is perhaps so under some circumstances in the higher vertebrata.

Theories of Cell Migration.—Cohnheim explained the passage of leucocytes by their spontaneous amœboid movements. The retardation of the blood current, which is itself a consequence of arterial dilatation, causes them to collect at the sides of the vessels, where they lose their spherical form and become flattened against the wall, and at the same time commence those movements, consisting in the protrusion of protoplasmic processes, which they exhibit elsewhere. These processes, sent out in all directions, extend themselves where they meet with least resistance, through certain gaps or stomata which Cohnheim believes to exist in the veins between the epithelial cells of their inner membrane and in the capillaries between the similar cells which he believes to constitute their walls. The middle and outer coats of the veins are also supposed by him to possess interstices sufficient to allow the leucocytes to pass. The red corpuscles, which have no spontaneous movements, he supposes to be forced by mere increase of hydrostatic pressure through the openings, already enlarged by the passage of the leucocytes. On this view, then, the structure of the vessels remains normal, though possessing a natural adaptation for cell migration; while others, on the contrary, deny the existence of these openings in the vascular walls, and regard the permeability of the vessels to their contents as a morbid phenomenon, and the essential cause of exudation and cell migration.

According to Dr. Burdon Sanderson, the retardation of the blood current (which is the first stage of stasis), and the exudation of liquor sanguinis, as well as that of the white and red corpuscles, are all due to the same cause, namely, a "local change in the vascular walls." With respect to the nature of this change, he says that "The mode in which an injury changes the living substance of the vascular walls so as to make them permeable to the blood is unknown. The nature of the change itself is also unknown." From certain appearances observed in the formation of new vessels he is, however, led to infer "that the primary change consists in the transition of the material from the formed into the plastic condition; from a state in which it is resistant, because inactive, to one in which it is more living, and therefore more labile."

Theories of the Cause of Stasis.—Whether this phenomenon be regarded as the cause, the consequence, or the mere accompaniment of exudation, its causation is a question of much interest, and was described in the first edition of this work as the great and unsolved problem of inflammation. Henle regarded it as due to the inspissation of the blood plasma, resulting from exudation; this condition causing endosmotic changes in the red corpuscles, by which they are disposed to aggregate. Rokitansky suggested in addition the formation of fibrinous coagula. We do not doubt that the changes wrought by exudation, draining away of liq. sanguinis, multiplication of white corpuscles, and coagulation of the fibrine, may all exercise considerable influence in prolonging and confirming the stasis, and determining its results, but we do not think they are the primary and causative phenomena. It seems to us a very important fact, which Mr. Wharton Jones states, and which we have observed ourselves, that "stagnation commences in the capillaries, and extends from them to the veins on the one hand, and the arteries on the other." We have seen the blood stagnant in the capillaries, while it was moving on steadily through an adjacent artery and vein. This points to the capillaries as the part where the arrest commences. Again, it is a very important circumstance, that, if a strong stimulus be applied, the stasis takes place almost immediately: it seems as if the blood were suddenly coagulated in the capillaries. That in the healthy state the red corpuscles pass on so smoothly and uninterruptedly, within their even relatively narrow channels, that they shun, as is well known, the walls, and allow a thin layer of the fluid in which they float to intervene, while the colourless corpuscles show no such tendency, but affect a preference for the so-called still layer, and move slowly onwards in it; that there is decidedly a tendency of the red globules to aggregate together in blood drawn from the body, while this seems to be held in abeyance when the blood is within the vessels; that the smallest capillaries, according to Wharton Jones, are traversed chiefly by plasma, and by a few colourless corpuscles, one after the other, with only a single red corpuscle now and then; that the red and colourless corpuscles show no tendency to stick together,—appear to us very significant facts, which require some such hypothesis as that offered in Wharton Jones' report, April, 1844, viz.: "That there exists some sort of attraction between the colourless corpuscles and the walls of the vessels, but an absence of attraction, if not a repulsion, between the red corpuscles and their walls, as also between the red and colourless corpuscles."

Fig. 7.



Production of stasis from Mr. Wharton Jones' Essay, Guy's Hospital Reports, vol. vi. p. 35.

We have already (page 81) given reasons for rejecting the adhesiveness of the corpuscles themselves as the cause of stasis, and the determining cause must be one which acts by means of the vessels in which the blood is contained. It must, therefore, by some change either (1) in the walls of the vessels themselves, (2) in the nerves acting through the vessels, or (3) in the surrounding tissues, altering their normal vital relations to the blood. It must, in fine, depend upon some derangement of the normal play of forces between the vessels and their contents. Mr. Lister's* experiments, published since the first edition of this work, strongly support the views that the primary change is not in the blood. He attributes stasis to some influence of the tissues on the blood circulating in them, which brings it into the same condition, with respect to cohesiveness of its corpuscles, as that of blood drawn from the body. Mr. Wharton Jones formerly conceived that it was the nervous influence which prevented the red corpuscles from aggregating together within the vessels, as they do out of the body, and that stasis depended on the suspension of this influence. It was an ingenious and probable opinion, but is disproved by the experiments which he himself has performed, and which are related in the Astley Cooper Prize Essay.

All that can be said respecting the alterations in the vessels themselves, may be found in Dr. Sanderson's essay. With regard to *stasis*, he adopts the same explanation as with respect to exudation of serum and cells, believing it to be due not to the properties of the red corpuscles, but to an abnormal condition of the vessels; and quotes some experiments of Ryneck,† which showed that other fluids, such as milk, circulating in irritated vessels, are also liable to stagnation. Mr. Lister had previously shown that healthy blood stagnates in the vessels of a part exposed to the action of irritants. None of these experiments, however, show that the original change is not one outside the vessels, in the tissues, by which the vascular wall is secondarily affected.

Our limits forbid discussion; and we therefore simply pass on to state our own opinion, so far as we may venture to offer one, on this *quæstio vexata.* We saw reason to believe that the tissues, in virtue of their nutrition power, exercised an influence on the movement of the blood: that in active hyperæmia their attractive force was increased; and we would now add, that it is through the failure of this nutrition power that we believe stagnation takes place. The exact nature of the influence exercised by the tissues over the blood, which traverses the capillary channels, is unknown. All that we can discern is, that it is such as promotes its free passage through them; and therefore, when it is in abeyance or greatly altered, it is to be expected that the circulation will be interrupted also. More than this we cannot gather from the observed phenomena; and we would only offer the remark, in conclusion, that in coincidence with the establishment of complete stasis, cessation of the natural function of the part occurs, and other processes commence—the exudative, in which the plasma, that in a healthy state would have ministered to and maintained healthy action, is consumed in wasteful or even destructive changes. Whether the stasis depend solely on a persistence and exaggeration of the attraction of the tissues for the blood which exist in active hyperæmia, or upon this and an abolition of the natural nonaggregative tendency, or even repulsive tendency of the red corpuscles for each other and for the walls of the vessels, must remain uncertain; but the coincidence before noticed must be allowed to give considerable support to the main point, on which we would insist, viz., that the nutrition power of the tissues is chiefly concerned in the production of the flow of active hyperæmia, and the stasis of inflammation.

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* "Philosophical Transactions," 1858.

† Rollett's "Untersuchungen," Leipzig, 1870, p. 103.

Not very different from this, at least on this special point, is the theory of Virchow. He supposes the starting point in the chain of phenomena in inflammation to be in the tissues. The stimulus (whatever it may be) is supposed to act upon the cells, not directly on the vessels, arousing in them greater nutritive activity; in consequence of which there is more rapid exudation of nutrient material and increased afflux of blood. Thus are caused the phenomena of hyperæmia and exudation. We must now discuss the nature of these changes in the tissues, on which the theory of Virchow rests.

Alterations of the Tissues in Inflammation.—Beside the phenomena which have been described as occurring in the blood and the vessels, important alterations are found in tissues which have been the seat of inflammation. These changes are partly constructive, partly destructive; we shall first consider the former. Constructive or reproductive changes are shown by the presence in the tissues of a number of elements which were not there before, these elements not being specialized cells such as epithelial or connective tissue cells, but having an indeterminate or embryonic character. These may be summarily described as young cells. Three hypotheses have been made about the origin of these elements:—(1) that they are formed in an amorphous blastema exuded from the vessels; (2) that they result from the multiplication of the elements of the tissues; (3) that they are extravasated leucocytes. The same three hypotheses have been made respecting the origin of the corpuscles of pus and inflammatory lymph, with which, of course, the new elements of the tissues are identical. We are now concerned only with the second of these theories, in support of which it has been asserted that evidences of multiplication can be traced in the tissue-cells of an inflamed part. These evidences are such as the presence of two or more immature cells on the precise spot where only one is normally found; cells pressed closely together, and showing by their form that they have been derived from the division of one mass; cells containing several nuclei, &c. In vascular organs, it is difficult or impossible to distinguish leucocytes escaped from the vessels from cells which may be the brood of the tissue elements, and hence non-vascular tissues have been especially studied. The observations of Redfern on the ulceration of cartilages were among the earliest and the best. In this condition, the gradual change of a single cell into a collection of pus cells may be traced within different cartilage capsules. This process was formerly not regarded as inflammation, but similar phenomena have lately been observed as the consequence of artificial irritation of cartilage.* The favourite object of investigation has been, however, the cornea, both on account of the simplicity of its structure and the clearness with which its elements can be brought into view by certain re-agents. By inducing inflammation with nitrate of silver, then excising the cornea, and treating it with solutions of gold, many of the connective tissue corpuscles are seen to have undergone changes indicative of increased vitality and proliferation; though there are always some which exhibit no change. In those which are altered the rays or processes (of the stellate corpuscles) show signs of contractility; then they become thicker and shorter, till they become converted into clumps of irregular shape; some merely granular, others containing newly-formed nuclei. They also show amœboid movements, the nuclei changing their relative position, as granules do in the body of an amœba. These changes can only be clearly seen within the first twenty-four hours after the application of the stimulus; as after this the cornea becomes turbid with a vast number of leucocytes, of which most (or, according to Cohnheim, all) are emigrants from the blood-vessels. Dr. Burdon Sanderson, from whom the above account is taken, thinks "it cannot be doubted that the little spheroids contained in the amœba-like masses are young pus corpuscles; and that even in the cornea suppuration must be regarded as, at all events, in part, a process of germination." These conclusions, first announced by Recklinghausen, before the publication of Cohnheim's researches, are denied by the latter pathologist, who endeavoured, by injecting finely divided pigment into the veins, to mark the blood corpuscles, and thus distinguish them when emigrated, from the elements of the tissues. Leucocytes thus coloured were found in the inflamed cornea, and thus plainly proclaimed their origin from the blood. But according to other observers *all* the leucocytes seen are not thus coloured.

The same difference of opinion exists with respect to another object studied in the living state, namely, the frog's tongue, in which the observations of Cohnheim and Stricker are precisely contradictory. The same is true of the omentum, with respect to which Cohnheim asserts that the inflammatory cells are merely superimposed upon the unchanged elements of the tissue, while most other observers have traced the formation of pus, or inflammatory cells, from the serous epithelium, or from the connective tissue nuclei. The annexed figure shows the actual appearance of an inflamed omentum, and exhibits the germination of the endothelial elements. It should, however, be said that such germinative changes are, according to the editor's observations, of normal occurrence in the omentum, and only appear to be more abundant and active in inflammation or other hyperæmic conditions. Another possible source of new elements should be borne in mind, namely, the multiplication of leucocytes after they have escaped from the vessels. That this is possible is shown by the observation of Klein, who was fortunate enough to see a leucocyte divide on the stage of the microscope; and Recklinghausen, some years ago, concluded from observations made on an excised cornea maintained in a living state, that new cells were produced by the multiplication of the unfixed corpuscles of the cornea or free leucocytes.

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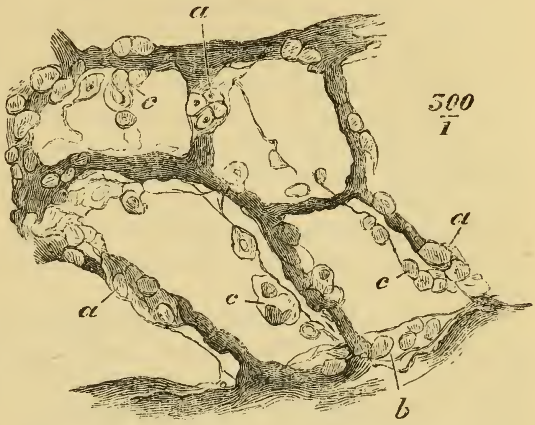
* Kremiansky, "Wiener Medizinische Wochenschrift": 1868. Cornil et Ranvier, "Manuel d'Histologie Pathologique," p. 72. Paris, 1869.

It is clear that the evidence is strongly in favour of the occurrence of changes in the tissues, denoted by increase and proliferation, but it may be still a question whether these changes are a consequence of the nutritive stimulus given by hyperæmia and exudation, or are themselves the original starting point of the inflammation, and the cause of the increased flow of blood and exudation of serum. The special forms and consequences of inflammatory new growth will be considered afterwards.

FIG. 8.



Fenestrated portion of the human omentum in acute peritonitis, caused by ruptured intestine. (From a preparation by the editor.) *a a a*, Groups of nuclei, or young cells, produced by proliferation. *b*, Large mother-cell, or endothelial plate, containing many nuclei. *c c c*, Free corpuscles, or young cells, entangled in threads of fibrin. These may be either new elements produced by germination, or emigrants from the blood-vessels.

VARIETIES OF INFLAMMATION.

These depend partly on differences inherent in the subject, partly on different exciting causes. What is called *Sthenic* inflammation is that which occurs in a healthy person, from exposure to cold, or the application of some irritant which does not contaminate and depress the general system. The symptoms, if the inflammation is extensive, run high, the febrile excitement is considerable, the pulse of good force, but moderate frequency, and depressant remedies are borne well. The exudations contain much plastic matter, and pus, if formed, is of the kind termed *laudable*. *Asthenic* inflammation occurs in persons originally weakly, or rendered so by the action of the exciting cause, as in the inflammations of influenza; the pulse, though frequent, has no strength, the fever is of a lower type, and the effused matters manifest little plasticity. The terms *acute*, *sub-acute*, and *chronic*, have reference mainly to the periods of duration of the inflammation, or to the rapidity or slowness of its course. Acute inflammation is often, but not necessarily, sthenic; chronic and sub-acute are often not asthenic. Many changes are commonly said to proceed from chronic inflammation, which probably belong more to the class of degenerations; it is, however, difficult to draw any marked line between the two. The term "sthenic" has been objected to by some who have taken it to imply that the morbid process was attended by an increase of vital force. Such of course is not the case; but the name may be well retained, to designate a variety of inflammation, which differs in several respects, notably in the σθένος of the pulse from another form, whose features are those of debility.

Intensity of Inflammation.—Inflammations may be of very different grades of *intensity*, as is well exemplified in the case of the pleuræ. Some pleurisies give rise to effusions which are scarcely more than modified liquor sanguinis, and are easily resorbed, while others fill the serous cavity with perfect pus, and depress the vital powers seriously. It is important to be aware that an inflammation may be sub-acute or chronic from the outset; an acute inflammation cannot well be overlooked—a chronic, if unpreceded by acute symptoms, may easily be: such inflammations are sometimes said to be *latent*. Congestive inflammation differs not much from asthenic; its effusions are of the same kind, but it partakes in a considerable degree of the nature of passive hyperæmia—indeed, is such originally, and has, subsequently, inflammation, acute or subacute, grafted upon it. This must be remembered in treating it.

Specific Inflammations.—The chief character of erythematous or erysipelatous inflammation is its tendency to spread and travel over an extensive surface; this seems to depend, at least in part, on the peculiar character of the effusion, which consists, for the most part, of serum, or sero-purulent matter, and not of fibrine, which in phlegmonous inflammations establishes a barrier between them and the surrounding textures. The general symptoms are in most cases those of adynamic or typhoid fever; depletion is injurious, and stimulants are required at an early period. There is good evidence to show that a peculiar poison, capable of being communicated by infection, is the cause of these inflammations, and that this acts upon and modifies the system, even before the phlogistic process has made its appearance. They are, therefore, with respect to their exciting cause, to be ranked together with other inflammations, such as the rheumatic, gouty, syphilitic, &c., each of which manifests certain peculiarities, but depends, essentially, upon the presence of some *materies morbi* in the blood. These inflammations also exhibit very clearly the affinity of certain parts of the tissues for certain morbid matters, which are their exciting causes; thus, lepra has its seat of election about the prominence of the knees and elbows; eczema prefers the side of flexion of the limbs and the bends of joints; lichen affects the outer sides. The same is exemplified in the action of many medicines and poisons; arsenic, in small doses, produces conjunctivitis—in larger, inflammation of the stomach and intestines, corrosive sublimate inflames the larger intestines, mercury the gums, and so on; in all these cases the affinity or attraction of the elements of the tissue for the substance is clearly evinced. *Diphtheritic* inflammation is characterized by the early exudation upon mucous surfaces of a film or membrane of fibrinous matter of dirty white or greyish appearance; this may extend over a considerable tract, commencing often in the fauces, and thence spreading to the mouth, the larynx, the air passages, the œsophagus, and more or less of the alimentary canal. The subjacent mucous membrane is but little swollen, of a deep dull red, and inclined to bleed, on the removal of the exudation. The attendant fever is of a low kind, and much of the danger depends upon the insidious, almost latent manner, in which the exudation takes place, so that suffocation may be actually threatened before alarm is taken. This kind of inflammation occurs most often in epidemics. It has been observed in France that during its prevalence wounds and ulcerations assumed an unhealthy character, and were indisposed to heal. The anatomical characters of diphtheritic false membranes are very peculiar, and will be further considered hereafter. Of a somewhat similar kind are the croupous inflammations; among these we should, perhaps, rank the so-called bronchial polypi, the pieces of membrane which are passed after the irritation of calomel, and in some other cases of intestinal disorder, and, probably, some of the membranous exudations of dysmenorrhœa. *Hæmorrhagic* inflammation is another variety; it seems chiefly to occur in individuals predisposed to hæmorrhage, or in places where scurvy is prevalent. Dr. Williams has found it associated with cirrhosis of the liver and granular degeneration of the kidney. His opinion is, that it is more dependent on an altered condition of the colouring matter than on a deficiency of the fibrine. Its character is decidedly asthenic.

Scrofulous Inflammation.—Scrofulous inflammation is not so much a distinct variety as some others, but has been distinguished as follows:—

The inflammations called scrofulous occur almost exclusively in children and young persons, especially such as have what must be called a special tendency or diathesis. In such persons a slight irritation will produce an inflammation of singular obstinacy, and extremely liable to cause permanent destruction or injury of parts. Their tissues seem to have less power of resistance to disease, and less power of restoration than those of "healthy" persons; and hence the inflammations partake of the same character. Scrofulous inflammations are also very liable to affect secondarily lymphatic glands, and excite in them the same lingering and destructive inflammations. The products of inflammation are not absorbed so completely as in normal inflammations, and thus produce the cheesy masses often called scrofulous matter; but this is seen also in other inflammations. The changes, inflammatory and otherwise, which accompany (either as cause or effect) the presence of tubercles, have the same general character as those originally called scrofulous. To sum up the views of Virchow (which we have followed), scrofulous inflammations are distinguished by their physiological character, as being easily aroused, obstinate, and destructive; by their anatomical relations, being often connected with the lymphatic system; and by the tendency to generate persistent masses of cheesy or degenerated matter.

CAUSES OF INFLAMMATION.

We have already alluded to the *causes* of inflammation in the foregoing remarks, and shall now do little more than briefly enumerate them. Predisposing causes are almost always debilitating influences; a strong part is less liable to inflame than a weak one—a previous attack of inflammation especially renders a part more prone to undergo a second. Certain unhealthy conditions of the blood (of which that induced by foul air is one) predispose the system to inflammation from trifling causes, which would pass inoperative in a sound state. Exciting causes are either such as act on the part directly which they inflame, or indirectly through the medium of another.

Direct Causes.—These may be *mechanical* irritants, such as a splinter in the flesh; or *chemical*, as a strong acid, or acrid salt; or *vital*, such as mustard, &c., whose operation only affects living structures. It is remarkable that the urinary and biliary secretions which excite only healthy action in the mucous surface over which they naturally flow, and which, or at least some of their constituents, produce no particular injurious effects when absorbed into the blood, act as the most violent and fatal irritants upon serous membranes, and the areolar tissue, when effused into them. This shows clearly the important part played by the tissues themselves in the process of inflammation: that which is a healthy stimulant to one texture is the cause of destructive inflammation to another. The production of local inflammations from the presence of some substance in the general mass of blood, for which certain parts seem to have a special affinity, has already been noticed, but we may add, that it is in these cases that we observe the interesting phenomena of symmetrical disease;* the corresponding parts of the two lateral halves of the body being affected almost to the exclusion of others. Here, again, we have evidence of the predominant influence of the tissues; the parts which are exactly alike are affected alike, and the *materies morbi* passes by others. How unable is the neuro-pathological theory to explain such instances of inflammation !

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* For a most interesting exposition of the subject of symmetrical diseases, we refer to Mr. Paget's Lectures for 1847.

Indirect Causes.—The second class of causes are those which act indirectly on the part which suffers. The most common of these is cold, which appears to act by repelling the blood from the surface, and causing it to accumulate in some internal part. This will be different, according to the previous predisposition ; thus one person, as the result of a severe chill, will have bronchitis, another diarrhœa, a third peritonitis, a fourth renal congestion, and so on. Malaria, the repelling of eruptions, the arrest of habitual discharges, the sudden healing of ulcers, are also recognized as causes of internal inflammations, which they probably produce in the same way as cold, but the active congestion of the incipient process is more apt to issue in hæmorrhage. It is matter of much uncertainty as to how many inflammations originate ; they come on, as it seems, spontaneously, without the individual being aware of any exciting cause. Both as respects these, and those which are produced by cold, &c., it seems to us necessary to recognize some special condition of the tissue, which in the one case converts the congestion into an inflammation, and in the other is the sole and efficient cause. In ague, for instance, during the cold stage of each paroxysm, considerable congestion of the internal viscera takes place, but inflammation of these is comparatively rare. In healthy states of the system, the surface may remain severely chilled for several hours, during which the blood must accumulate in the internal organs, but this does not occasion inflammation. These instances show that mere repellent influences producing congestion are not adequate to produce inflammation solely by themselves. Even in the case of a common catarrh, there is much reason to believe that the inflammation of the mucous surface depends much more on some infecting miasm, as that of influenza, or some unhealthy condition of the blood, than on exposure to cold or wet ; and, in fact, many a cold occurs without any such exposure. In such cases the predisposing cause becomes the most important, or may even be the exciting also.

Influence of the Nerves.—We have not yet noticed the nervous influence in its relation to the causes of inflammation. This is considered the prime mover in the process by those who adopt the neuro-pathological theory ; in our view it holds a very secondary place. That disturbance of the nervous force *may* prove a cause of inflammation cannot be doubted ; Lallemand (quoted by Dr. Williams) "relates a case in which a ligature, involving the right brachial plexus, was followed by inflammation and suppuration of the opposite hemisphere of the brain." Sir J. Paget mentions a case in which a portion of a calculus imparted in the urethra excited inflammation, with deposits of lymph and pus in the testicle. Instances of a similar kind, or of active hyperæmia similarly occasioned, are not very rare, and they certainly prove the capability of the nervous influence to set on foot the inflammatory process. We have already given some evidence that it is not through this channel that the causes of inflammation usually operate; but certain special cases of the connection of nerves with inflammation may be noticed. In the disease called herpes zoster, discrete inflammation of the skin is met with over the distribution of a certain nerve, and is doubtless connected with some abnormality of innervation. The same is true of the skin disease called morphœa, or a form of scleroderma. Various changes, such as œdema, hyperæmia, &c., are observed as the consequence of injury or division of nerve trunks by wounds; but rarely true inflammation.

Summary.—Coinciding, as we completely do, with Sir J. Paget in his enumeration of four conditions as necessary to healthy nutrition, and believing that the derangement of one of these primarily, with secondary derangement of the others, occurs in every case of inflammation, we might divide the various causes which we have noticed into such as affect—(1) the contractility of the vessels; (2) the healthy crasis of the blood; (3) the nervous influence; (4) the life and nutritive actions of the part.

TERMINATION OF INFLAMMATION.

In strict language, there is only one termination to the inflammatory process, viz., that which is commonly called resolution, in which the diseased action ceases to advance, and then recedes by the same steps as those by which it arrived at the condition of stasis. The microscopically visible phenomena have been before described, and they correspond to the subsidence of the general symptom, to the paling of the redness, the lowering of the temperature, the lessening of the swelling and pain. The recovery of the part may be complete; but more often some, it may be slight, indications remain, for a time, of the by-past malady, and of some deficiency of the vital powers. These consist, in some degree, of congestion of the vessels, especially of the veins, from an enfeebled state of their contractility, in a less perfect fulfilment of the function of the part, and in a proneness to relapse on the application of slight exciting causes.

Inflammations which arise in consequence of a mal-crasis of the blood rarely undergo resolution, or if they do it is only to reappear in another part, and perhaps a more important. This constitutes metastasis. A good example of this transfer of inflammation from one part to another is afforded by some cases of rheumatism, and occasionally by the disease called mumps. As long as the *materies morbi* continues to circulate in the blood, it will tend to excite inflammation in one part or other: the best thing that can happen is, that it should locate itself in a part where it can produce no serious effects from interference with important functions, and there remain until the dyscrasia is at an end.

One important remark of Rokitansky must not be omitted, viz., that even resolution does not, especially if the inflammation has been extensive, leave the system in as favourable a condition as before the attack, since a large quantity of liquor sanguinis, which, during the stasis, has undergone certain changes, is set free to mingle with the general mass of the blood. This must produce a contaminating effect until it be eliminated. Hence the benefit of a free action of the skin, of a free flow of urine, purging, &c.

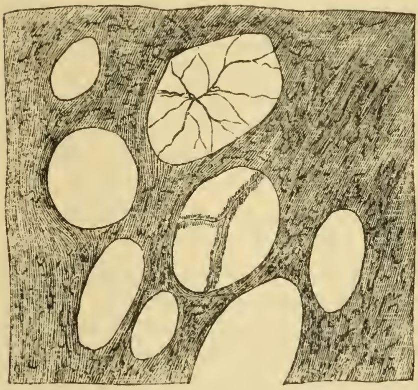
THE PRODUCTS AND RESULTS OF INFLAMMATION.

We now come to the consideration of, first, inflammatory exudations, and secondly, of the changes that take place in the tissues affected by them. The fluid effused in inflammation is commonly the liquor sanguinis, more or less modified, especially containing a less proportion of albumen and fibrine. An analysis by Simon, of fluid obtained by paracentesis thoracis, shows, in 1,000 parts, 934·72 of water, 1·02 of fibrine, 1·05 of fat, 48·86 of albumen and albuminate of soda, 11·99 of extractive matter, and 9·05 of fixed salts. The quantity of fibrine varies considerably. Some effusions consist chiefly of it, others contain very little; generally it may be said its quantity is in proportion to the vigour of the system, and the acuteness and sthenic character of the inflammation. The fibrine may coagulate quickly, or remain in its fluid state, for a length of time, in the part where it is effused. Blood corpuscles, or dissolved hæmatine, may be mingled with the exuded matter in various proportions. Though we often speak of serous effusions as the result of inflammations, yet the opinion expressed by Sir J. Paget is probably correct, that "an effusion of serum alone is a rare effect of inflammation, and that generally it is characteristic of only the lowest degrees of the disease." He mentions as instances inflammatory œdema of the mucous folds above the glottis, chemosis of the conjunctiva, and some forms of hydrocephalus. The fluid obtained from blisters contains either distinct fibrinous coagula, or only a small proportion of fibrine, together with multitudes of puriform corpuscles.

Varieties of Inflammatory Products.—The chemical examination of inflammatory products is very difficult, partly in consequence of the impossibility of procuring more than very small quantities, partly because they can so seldom be obtained pure; and almost the whole of our knowledge, therefore, has reference to the differences which are perceptible to the eye and to the microscope in the solidified and shaped constituents of the exudation. These, however, afford very valuable indications for forming a judgment of the nature and tendency of the process from which they spring; the most important differences between different exudations being the greater or less proportion of cells to the other constituents. (Theories as to the origin of these cells we have already alluded to, and shall speak of again.)

Thus Sir James Paget distinguishes two types of lymph, the fibrinous and corpuscular, which agree generally with what Rokitansky calls fibrinous and croupous; the latter being that in which the cells predominate. These cells, called at different times lymph corpuscles, exudation corpuscles, plastic corpuscles, and embryonic cells, are really identical with the corpuscles of pus, and cannot be distinguished from the white cells of the blood. Rokitansky* remarks that, as regards the abundance of these elements, exudations form a continuous series, from one with a minimum of them, to that form which is distinguished by a luxuriant production of them, and is called *pus*. These differences have often been brought into connection with differences of bodily constitution. Sir James Paget† found in the fluid derived from blisters in different persons very varying amounts of corpuscular elements, the number being generally in inverse proportion to their health and strength. These variations he attributes to differences in the condition of the blood. Recent discoveries have led some rather to attribute them to variations in the facility with which blood corpuscles can leave the vessels, and these in all probability to differences in the structure of the vascular walls. This view, however, cannot explain the difference between the basis-substance of different specimens, which is made up of fibres in some and granular matter in others. Another variety of exudation, the *hæmorrhagic*, is distinguished by the abundance of red corpuscles, though these, as we have seen, generally pass out of the vessels in small number. It has been thought that blood pigment sometimes transudes without the corpuscles, but this has not been clearly proved.

Fig. 9.



Fibrinous exudation on pleura in process of absorption: areolæ form in it, and reduce it to filamentous bands.

SUPPURATION.

We have next to consider that form of exudation in which the cells preponderate enormously, and in which the noncellular part is fluid, viz., pus, which, in the words of Rokitansky (loc. cit.), has been till now separated in an unnaturally sharp manner from other exudations.

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* "Lehrbuch der Path. Anat.," third edition, vol. i., p. 136.

† "Lectures on Surgical Pathology," second edition, p. 252.

There are many varieties of *pus;* but that which is commonly called healthy (laudable) is that which we shall take for a typical description. It appears to the naked eye as a creamy, thick, opaque, and homogeneous fluid; communicates an unctuous feeling when rubbed between the fingers; is of a yellow or whity-yellow tint; sweetish or insipid; and, while warm, gives off a peculiar, mawkish smell. Its specific gravity is 1·030–1·033. If allowed to stand some time in a tall, narrow glass, the fluid separates into a thickish *sediment*, more or less abundant, and a supernatant *serum*. This serum, according to Vogel (whose account we shall use freely) is identical with the serum of the blood, containing one to four per cent. of albumen, extractive and saline matters, and fat. It coagulates when treated. The reaction is alkaline; but it readily becomes acid, from the generation of an acid, which is commonly supposed to be the lactic. A peculiar substance, called pyin by Güterbock, somewhat resembling mucin, was formerly thought characteristic of this pathological product, but is not usually found in normal pus. It is an albuminoid substance, containing a larger proportion of oxygen than the true albuminates.

Chemical Constitution of Pus.—The following analysis, by Dr. Wright, exhibits the main features of the chemical constitution of pus very well. They apply, of course, to pus as a whole, not to the serum only. The large quantity of fat in pus is remarkable, as well as the amount of albumen—the latter sometimes exceeding that contained in the liquor sanguinis.

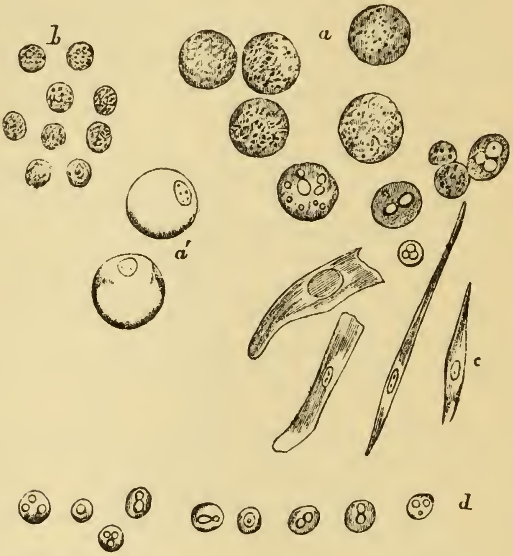
	From a vomica.	From Psoas Abscess.	From Mammary abscess.
Water	894·4	885·2	879·4
Fatty matter	17·5	28·8	26·5
Cholesterine	5·4		
Mucus	11·2	6·1	—
Albumen	68·5	63·7	83·6
Lactates, carbonates, sulphates and phosphates of soda, potash and lime	9·7	13·5	8·9
Iron	A trace.	—	—
Loss	3·3	2·7	1·6

The following analyses are from German sources:—

	Bibra. I.	Bibra. II.	Bibra. III.	Bödeker.
Water	907	862	769	887·6
Pus-cells and mucus	—	—	—	46·5
Albumen	63	91	180	43·8
Extractive matter	20	29	19	—
Cholesterine and fat	9	12	24	10·9
Salts	6	9	9	—
Sodium Chloride	—	—	—	5·9
Other alkaline salts	—	—	—	3·2
Earthy phosphates & iron	—	—	—	2·1

Serum of Pus.—The serum resembles blood serum in most of its properties. It contains paraglobulin, or fibrino-plastic substance; albuminate of potash (which is identical with casein), and by coagulating at about 75° C., shows that it contains ordinary serum albumen. It also contains, according to Hoppe, myosin. The following substances have also been detected in pus, but cannot be referred specially to the serum or corpuscles:—Protagon, chondrine, and gelatine; various fatty substances, as cholesterine, free fatty acids, soaps (alkaline salts of fatty acids), leucin, tyrosin, and xanthin (nitrogenous derivatives of the albuminates). Occasional or abnormal constituents are bile acids and bile pigment (in jaundice) and sugar (in diabetes). Under certain circumstances pus assumes a blue colour, which will also affect the bandages used for wounds. This has been shown to depend upon the presence of a peculiar vibrio which lives on the surfaces of wounds and the bandages. The colouring matter can, however, be separated, and obtained in blue crystals; it is called *Pyocyanin*. The ash of pus amounts to five or six per cent., and has a similar composition to the ash of blood; but the ash of the serum alone contains more sodium chloride than that of blood serum (seventy-two per cent.), and also more potassium.

Fig. 10.



Corpuscles from a pustule.

a, Large granulous exudation globules. *b*, Pus corpuscles. *c*, Nucleated fibres. *d*, Pus corpuscles, their nuclei brought into view by acetic acid. *a′*, Granular exudation globules, their nuclei brought into view by the action of water.

Corpuscles of Pus.—The *sediment* consists almost entirely of small organized bodies, the well-known pus-corpuscles, or pus-cells. These are, when dead, of spherical form, about $\frac{1}{3000}$ inch in diameter, formed of a mass of soft granulous substance, enclosing a varying number of nuclei, about $\frac{1}{6000}$ inch in diameter. These are, in well-formed pus cells, for the most part concealed by the surrounding substance; but in some cells, even of healthy pus, and in all those of pus of an inferior kind, they are easily perceptible, even without the aid of acetic acid. Occasionally a single nucleus exists; but more commonly it is made up of two, three, four, or even five large granules. The single nuclei are always the largest, and it is generally held that the multiple nuclei are formed by a process of division from the single nucleus. When seen in the living state the form of pus-corpuscles is eminently variable; they show the amœboid movements and changes of form characteristic of masses of living protoplasm, and may be seen continually to push out and retract prolongations of their substance. Molecular movements may also be seen in their interior, like those in salivary corpuscles. The amœboid movements are said by Recklinghausen to be the more active, the more recent and severe the inflammation. They may be seen in pus from granulating surfaces, from inflamed mucous membranes, pustules, &c., if it be slightly diluted with a half per cent. solution of sodium chloride, or better, with some natural serous fluid. The movements are stopped by concentrated saline solutions, by water, and by the pressure of a cover glass, or instantly by exposure to a high temperature. They gradually cease in a cold atmosphere, but may, up to a certain time, be restored by heat. Most cells, from large collections of pus, are, however, already dead, and show no spontaneous movements. They often show also a sort of envelope or cell wall, formed by coagulation of the protoplasm, but a single pus-cell cannot, as a rule, be distinguished from a lymph or white blood-cell.

Effect of Reagents on the Corpuscles.—When already dead and motionless the pus globule is not very remarkably affected by being placed in water. It becomes somewhat swollen, and more spherical—and the nucleus becomes somewhat more apparent, but it is not destroyed and burst so rapidly as the blood globule. In blood, urine, mucus, saliva, it is unaltered. Acetic acid renders the granulous contents translucent, and brings out the nucleus more definitely. It renders the envelope also more transparent, but does not destroy it. Other dilute acids have a similar effect. Caustic and carbonated alkalies, and borax convert the whole corpuscle into a viscid mass, leaving only very minute dark molecules, whose import is uncertain. Pus, Dr. Walshe says, possesses a remarkable power of resisting decomposition. At the end of months some corpuscles may still be found unchanged, among others that are dissolved. It even retards the putrefaction of substances which are placed in it; but at the same time seems to exert upon them a corrosive influence. Pieces of flesh put into fresh pus gradually lost weight, and were at last dissolved, without any evidence of putrefaction having occurred. Under similar circumstances Recklinghausen found that pieces of coagulated albumen became infiltrated with pus-cells, which had penetrated their substance. Mr. H. Lee has shown that pus possesses a remarkable power of accelerating the coagulation of blood. In one experiment the blood, which had pus (healthy) added to it, coagulated in six minutes; while that which was left by itself required twelve. This is probably due to the paraglobulin or fibrino-plastic substance which has been found in the serum of pus, and which reacts with the superabundant fibrinogenous substance of the blood serum. Much weight has been laid on this property of pus, in relation to the theory of pyæmia.

Formation of Pus Corpuscles.—With respect to the origin of pus corpuscles there is the same difference of opinion as exists about the corpuscles of inflammatory lymph already spoken of; and there can be no doubt that the explanation of the one will also be the explanation of the other. It was at one time pretty generally held that pus-corpuscles were formed in a fluid blastema, by the process of "free cell formation," either by the grouping of granules or by the growth and enlargement of minute granular globules. In some cases also it was held, from a solid blastema of coagulated fibrine, by the liquefaction of the solid fibrine, and the multiplication of the corpuscles (which when very numerous, are called pus-corpuscles); or by a similar process, from granulation tissue; so that, in Paget's words, "the cells of pus from wounds are ill-developed or degenerate granulation cells;" and "the most frequent degeneration of inflammatory lymph is into pus." In distinction to all these views came the comprehensive generalization of the Cellular Pathology, which uniting in one scheme the cells of ordinary inflammatory lymph, of granulations, and of pus, regarded them all as derivatives of the cells or nuclei of the tissues. At first the power of producing new cells was supposed to be limited to the connective tissue, but it was afterwards shown that it must, on the same grounds, be ascribed also to epithelial structures. This theory was for a long time accepted universally in Germany and by many pathologists in this country and in France; but it has since the researches of Cohnheim, already mentioned, undergone much curtailment; and there are probably few observers who do not admit that many at least of the pus-corpuscles are nothing but emigrant blood cells. Without entering much into details, we must look for a moment at the explanation given of these two theories of suppuration in solid organs or abscess, and suppuration on mucous surfaces.

Abscess.—The changes of tissue elements, in the formation of an abscess, or in purulent infiltration, were studied with extraordinary minuteness by Weber, in 1858.* He found in all the surrounding parts those appearances which are regarded as indicating proliferation of the tissue elements; such as cells with several nuclei, groups of cells occupying the place of one; cells within others; and so on. These appearances were principally seen in the connective tissue corpuscles, but also in the nuclei of the sarcolemma of muscular tissue; the neurilemma of nerves, and the various supporting structures of glandular organs; and in addition, as pointed out by other observers, the nuclei of the blood-vessels. In consequence of these processes the interstices of organs become filled with young cells, which are in fact pus-cells, and the result is *purulent infiltration*. Simultaneously with the changes already described the non-cellular or inter-cellular part of the tissues undergoes important changes. The albuminous constituents become in part replaced by fat, the solid parts soften and liquefy; the whole passes through degenerative changes, ending in destruction; so that in place of the normal tissue there is a collection of newly formed pus-cells, in fact an abscess. What the cause of this destruction of tissue is, whether it is due to pressure of the newly formed cells, to some peculiar solvent action, or to simple interference with normal nutrition, is not certainly known. But it is plain that these destructive properties, whatever they are, must cause an abscess, once formed, to increase in the manner in which we know that it does.

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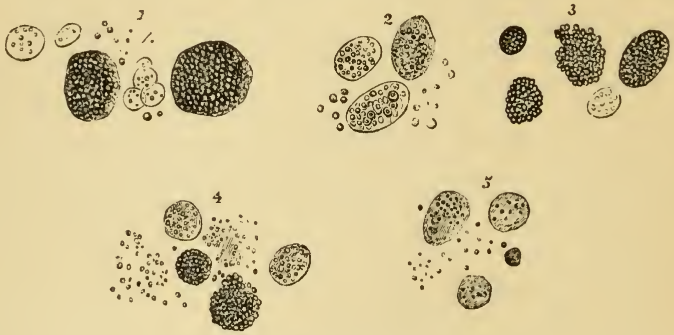
* Virchow's "Archiv," vol. xv.

On the migration theory of Cohnheim the collections of pus-cells are of course regarded as escaped leucocytes, which will tend to move into places already occupied by collections of pus, because this will be, on account of the destruction of the tissue, the direction of least resistance. In the same way, by the active locomotive properties of pus-cells, is explained the possibility of *drawing* an abscess to the surface (by poultices, &c.); the application of moist heat rendering the tissues more permeable to active elements. The enlargement of an abscess once formed is explicable on the same grounds; and also through its wall (the pyogenic membrane) being, according to Burdon Sanderson, a structure which facilitates the extrusion of leucocytes.

Suppuration on Mucous Surfaces.—The production of pus on surfaces covered with epithelium was first explained by Virchow as proliferation from the sub-mucous connective tissue, the newly formed elements making their way to the surface through the epithelium. Susequently it was shown that new elements might also be produced from the epithelial cells, the same changes indicative of proliferation being observed in these as are observed in connective tissue corpuscles. The fact that cells, resembling pus-corpuscles, are seen within epithelial cells cannot be doubted, though it is otherwise explained on the theory of Cohnheim. According to this observer the epithelial elements, like those of the connective tissue, are perfectly passive; all the new formed cells are emigrants from the vessels, and make their way through the superjacent tissues to the surface. Some of these penetrating into the substance of the epithelial cells appear as if they had originated there. This explanation is supported by the most recent observers, among whom may be mentioned Bizzozero.*

In suppuration of *serous membranes* it has been asserted, and also denied that the pus-cells arise from proliferation of the serous epithelial cells (or endothelia). The same is true of the connective tissue cells.

Fig. 11



Glomeruli and granular cells.

1, From ovarian cyst. 2, From cancer of breast. 3, From inflamed lung. 4, From inflamed pia mater. 5, From a case of tuberculous meningitis. The opaque cells are the glomeruli, the more simply granulous are the granular cells.

In the *nervous centres* the origin of pus-cells has been referred to the elements of the neuroglia, or connective tissue stroma.

In *glandular organs* they have been traced both to the glandular secreting cells and to the fibrous stroma.

In connective tissue structures, such as *cartilage*, *bone*, *tendon*, their proper cells, and in the cornea the branched corneal corpuscles have had the same function assigned them. In the skin suppuration has been referred both to the deep layers of the epidermis and to the connective tissue of the true skin.

Other Constituents of Pus.—The formed elements contained in pus, beside pus-cells, are free nuclei, derived from the disintegration of cells, red blood-corpuscles, and often what are called glomeruli or granule-cells. There are also many fatty molecules.

The granule-cell, exudation globule, or glomerulus, which was first described by Gluge, and called by him the compound inflammatory globule, is very frequently present in exudation, and is, speaking generally, a valuable sign of the existence of the inflammatory process, but not an infallible one. The granule-cell is usually of large size, from $\frac{1}{2400}$ — $\frac{1}{960}$ inch, mostly spherical, but often oblong, or of irregular shape. By transmitted light they appear dark, on account of their opacity; by direct, of a dead white. Their structure will be best understood from an account of their development.

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* Stricker's "Medizinische Jahrbücher," 1872.

It is now generally admitted that, as first shown by Reinhardt, granulous cells or glomeruli are the result of the transformation of other cells, either of normal or pathological formation. For instance, the cells of ordinary epithelium, and normal elements of the brain (probably those of the neuroglia), may undergo this change as well as mucus or pus-corpuscles, and the cells of any new growth, as cancer. The process consists, there is no doubt, in the formation of fatty molecules within the cell-protoplasm, and the gradual replacement of its albuminous by fatty material. This process is usually described as one of degeneration, and it is often induced by failure of nutrition, but recent observations on the early stages of inflammation in the brain (Stricker) show that deposition of fat must play some important part in the formation also of new elements. It is to be remarked, in conclusion, that a very abundant cell-growth commonly takes place in exudations, many particles of which are correctly denominated granular cells. These are not to be mistaken in descriptions for the *granule-cells*, into which they often undergo metamorphosis. The similarity of the name is unfortunate, but the subjoined sketch will make the distinction between the two very apparent. Moreover the granules contained in living cells (leucocytes, &c.) are in a great measure of albuminous composition, and dissolve in acetic acid, by which fatty granules are unaltered. The formation of granule-cells (glomeruli) in an exudation is a much less serious matter than the formation of pus. Inflamed lungs may contain myriads of the former and recover well, far better than if they were infiltrated with pus.

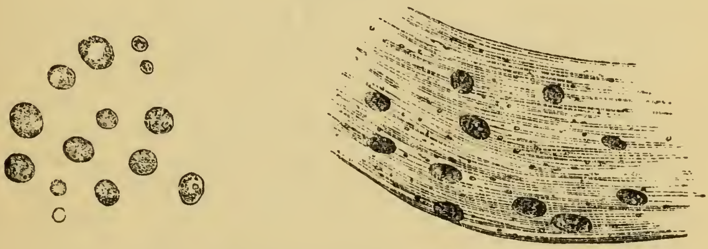
Varieties and Degenerations of Pus.—In various unhealthy states of the system pus is formed, which differs in several respects from that which we have now described. Small fibrinous flakes, epithelial particles, cholesterine scales, and prisms of triple phosphate may be mingled with it, as well as varying quantities of free oil. The pus-globules are in such cases often ill-shaped, feebly formed, conveying the idea of very defective formative power; the quantity of granular matter mingled with them is much increased.

Certain unhealthy kinds of pus, which Rokitansky comprises under the term (Jauche) sanies, are especially distinguished by their corrosive action upon the tissues, which he contrasts particularly with the bland quality of healthy pus. Their appearance is not at all constantly different from that of the normal fluid, but they are apt to be thinner, more tinged by hæmatine, of an offensive or ammoniacal smell, and to communicate a sensation of pricking or itching to the finger when applied to them. Their corpuscles are stunted, and their developments are variously altered, apparently by the "*gnawing*" action of the serum in which they float. It seems to us very questionable, whether the dissolving action which pus is said to exert on pieces of dead flesh belongs at all to it in a healthy and fresh state, and whether it does not really depend on the generation of acids within itself, in consequence of decomposition. Purulent effusions may degenerate into a semi-fluid amorphous mass, the corpuscles breaking up, and the serum undergoing chemical changes, often of a putrefactive kind. They may also undergo fatty degeneration, calcareous salts being liberated or deposited at the same time. Either of these two changes being premised, it is possible that a purulent collection may be absorbed, but it is only too probable that in the former case the result will be a fatal contamination of the blood by the decomposing matters taken up into it. Apparent temporary absorption may be easily produced by means which, creating a considerable demand for fluid, withdraw the serum from the pus-corpuscles; but as these retain their vitality, they soon attract a fresh quantity of blastema from the blood, and the abscess remains undiminished. This persistent vitality of the organized corpuscles of a fluid which is regarded as effete in the highest degree, and incapable of any further development, is certainly remarkable; one would rather have expected that they would have disintegrated rapidly.

Distinction of Pus.—Pus may be confounded with some other fluid, and the distinction is sometimes only to be made out by careful microscopical examination. What we have said respecting softened blood-clot, will show that a fluid having this origin may approach very closely to the purulent product of inflammation, so much so, that it may be doubted whether it be not in part identical with it. Since, as has been said, a single pus-corpuscle has nothing distinctive about it, the character of pus can only be given by the immense number and preponderance of such elements. Pus, when mixed with a fluid such as urine, may sometimes be distinguished from other substances containing similar corpuscles, as mucus, by the great abundance of fatty matter. From epithelial detritus and the broken-down contents of hydatid cysts the microscope readily distinguishes it.

Mucus.—The last inflammatory product which we have to notice is mucus. Speaking correctly, it is only unhealthy mucus which comes under this head: for it is perfectly clear that several internal membranes secrete a mucous fluid. The distinction between this and the morbid product is tolerably precise, and easy to be ascertained. The former is a tenacious, clear fluid, containing only some admixture of the epithelium of the membrane producing it, and having no special corpuscles of its own. The latter is loaded with corpuscles, identical with those of pus, together with a varying quantity of epithelial *débris*. Between such mucus and pus it is evident that a close analogy subsists. Mucus may be distinguished, like pus, into a fluid, the *liquor muci*, and corpuscles. The liquor muci, as we find it in the secretion of a membrane which has been subjected to moderate irritation, is a transparent, tenacious, more or less stringy fluid, of alkaline reaction, and more or less saline taste. The addition of acetic acid, or any weak acid, produces a kind of coagulation, and the formation of a granular precipitate, which Simon states is the mucine, the principal constituent of the fluid. It is insoluble in excess of acetic acid, which distinguishes it from albuminous substances. This is held in solution by means of an alkali, and consequently falls on the latter being taken up by an acid. Not much is known of this substance, except that it is a protein compound. Albumen or fibrine, treated with liquor potassæ, forms a transparent, viscous mass, having much resemblance to its solution. The proper corpuscles of morbid mucus are, as we are fully persuaded, and as the best observers state, quite identical with those of pus. They are usually mingled with epithelial particles, in very various stages of their formation, from a simple nucleus up to a complete cell. It is only in cases of prolonged and rather intense inflammation that traces of epithelium are wanting, and the so-called mucous corpuscles are crowded together, and seem to load the fluid. It is often very observable how the tenacious fluid, in which the corpuscles are entangled, in consequence of being dragged in one direction, produces an alteration of their shape; they thus become oval, or even staff-shaped. Granulous and oily matter is commonly diffused through the liquor muci, just as it is through that of pus. It is manifest from what has been stated, that the difference between mucus and pus consists essentially in the different nature of the fluids, not in that of their corpuscles. The old question, as to the means of distinguishing between pus and mucus, is manifestly of little moment, and has, in general, no interest for the practical physician. It is sufficient to state, that the liquor puris is albuminous, the liquor muci not so; that pus will mix with water, and mucus will not; that pus is dissolved, in some measure, by acetic acid, while mucus is coagulated; and that mucus generally contains traces of epithelium, while pus does not; while pus contains a very much larger quantity of fat.

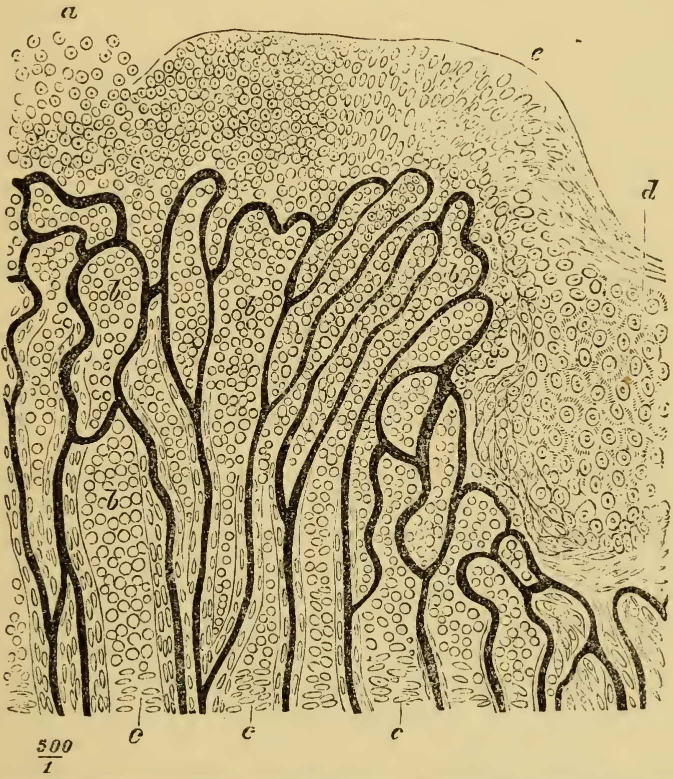
Fig. 12.



Separate corpuscles, and two blood globules.

Inflammatory New Growth. Granulations.—The products of inflammation already described are "effete," and serving no purpose in the organism are cast out of it; but inflammation also gives rise to a new growth which plays an important part in repair and healing of injuries, though itself transitory, and becoming always changed into something more highly organized. This is granulation tissue, which may be defined as a new growth proceeding from a wounded tissue, or from some part exposed by ulceration, and growing either on the external surface or within a cavity. The so-called granulations are characterized by a red colour, granular surface, and soft texture, and are very easily wounded. They are out-growths of the normal tissues, being continuous with them at the margin or base; while their connective tissue stroma and their blood-vessels are developments of the corresponding normal structures, and their cells formed by proliferation from normal cells. Granulations consist of blood-vessels, connective tissue, spindle-shaped or fibro-plastic cells, and round indifferent or embryonic cells. The characteristic cells are those called embryonic, since they resemble those of which immature organs in the embryo are composed. They are roundish, pretty uniform in size, from $\frac{1}{1800}$ to $\frac{1}{2500}$ inch, granular, and resemble generally those white blood cells which have a single nucleus. They differ from pus-cells chiefly in the nucleus. These cells are not absolutely in contact, but arranged with some intercellular substance, and a distinct *stroma*, so that the structure resembles that of a lymphatic gland, or what is called *adenoid*. There are also transitional forms between these and the spindle-shaped cells, which compose an immature connective tissue. There are also apparently transitional forms between the embryonic and epithelial cells, so that the embryonic tissue is of an indefinite type, which may be developed into other more special tissues. On the other hand, the granulation cells are sometimes not individually to be distinguished from pus-cells; and on the surface of ordinary granulations it appears as if many cells of the solid mass were constantly undergoing a retrograde change, and becoming converted into detached pus-cells. Granulations are richly supplied with blood-vessels, the walls of which are formed of spindle-shaped fibro-plastic cells (see Fig. 13), and are easily ruptured, which explains the well-known readiness of granulating surfaces to bleed. As the granulations grow, a new formation of vessels constantly takes place, apparently by out-growths from those already formed. The surface is always covered, more or less, with a greyish film of pus. This appears to be more or less copious as the growth tends rather to degenerative or to formative changes, the indifferent embryonic cells being capable of degradation into pus-cells or elevation into connective tissue elements. It is not certain that epithelial elements are ever formed from granulation tissue such as that here described; they being, as some think, never formed but from pre-existing epithelium. It is also possible that the pus-cells occurring on the surface of granulations may have merely escaped from the blood, the walls of the vessels, from their simple structure, favouring the escape of these elements. On the other hand, it is not certain that some at least of the true granulation cells are not escaped blood-cells. But no direct observations have been made on either of these points. Granulations, if healthy, tend to form connective tissue, which is of the kind called cicatricial, and is seldom very perfect, having a tendency to contract. This may, it is thought, become organized into higher grades of tissue; but the point is not quite clear. With regard to epithelium, it is not certain that this is ever formed from granulations; it is possible that it always arises from pre-existing epithelium. When "unhealthy," they tend to suppuration, and this must be regarded as a degeneration. Granulations, in fine, are the characteristic product of a productive or reconstructive inflammation, as pus is of a degenerative.

Fig. 13.



Section through the margin of a granulating surface in process of healing.

a, Production of pus. *b*, Granulation tissue with loops of blood vessels, the walls of which are composed of layers of cells. *c*, Commencement of the formation of cicatricial tissue containing elongated or fibro-plastic cells. *d*, Completed epithelium. *e*, Young epithelial cells. (After Rindfleisch.)

Changes in the Inflamed Parts.—We enumerate these as—(1) Enlargement; (2) Atrophy; (3) Ulceration; (4) Gangrene. The term "enlargement" is preferable to that of "hypertrophy," which is sometimes employed, because it conveys no such erroneous idea as that the part is truly increased in size by addition of more of its own proper substance, an occurrence which most rarely, if ever, is the result of any form of inflammation. The enlargement depends entirely on the infiltration of the tissue with some form of exudation-matter, which subsequently undergoes metamorphoses such as we have described, and is more or less completely absorbed. It often happens, however, that a part remains behind, and is converted into a low form of fibroid tissue, consisting of dense fibrous stroma, with many nuclei. This constitutes induration-matter, which resembles very much that which forms cicatrices; like which its tendency is to contract and shrink, thus compressing and obliterating the vessels of the part, and in this way, as well as by its pressure, inducing the atrophy of the tissues among which it is deposited. A good instance of primary enlargement and subsequent atrophy, resulting from inflammation, is afforded by some cases of cirrhosis of the liver. Other instances of enlargement are the thickening of bones, as a consequence of periostitis, and thickening of the skin in chronic eczema. Serous membranes also, are commonly thickened and complicated with fibrous adhesions, in consequence of attacks of inflammation.

Atrophy, the result of inflammation, is described by Rokitansky as depending upon the mechanical injury done to the tissues in the seat of inflammation by the exudation, as well as upon their being deprived by it of their proper amount of nutrition. Being thus rendered unfit for the discharge of their function, they fall to pieces, and are absorbed, together with the exudation. "This occurs with especial frequency in delicate, lacerable tissues, when large quantities of exudation have been effused, and such as are solid and capable only of slow reabsorption. Thus, in the inflammatory foci the substance of the brain, of the muscles, of the kidneys, &c., becomes lost, while there remains in its place one or more gaps, limited by cicatrix-tissue, which, if such gaps are small and numerous, causes a spongy, rarefied condition of the tissue."

Under the head of Atrophy we may include *softening*, which is a very usual result of inflammation, and declares, as its other phenomena do, its destructive nature. Hard bones, when inflamed, can be cut with a knife, inextensible ligaments yield and stretch, muscular walls of hollow organs are paralyzed, and dilatation takes place.

Ulceration implies that condition of a part in which more or less of its proper substance has become eroded, and has disappeared, in consequence of unhealthy action, so that a cavity remains. The term especially applies to epidermic and epithelial structures. This condition does not most commonly exist alone, but together with a greater or less amount of exudative and organizing processes. These are so far from being essential to it, that they constitute, in fact, the means by which its ravages are repaired; the formation of granulations, and the effusions of pus, are the characters, not of an extending, but of a healing ulcer. Instances of pure and simple ulceration are to be seen in the cornea, and in some ulcers of the walls of the stomach; they penetrate the tissue more or less deeply, so as sometimes to perforate it, without any surrounding thickening from the deposition of the lymph. In ulcers of the stomach, doubtless the corrosive action of the gastric fluid plays a part. When the erosion of the tissue goes on rapidly and extensively, forming a sore, with very irregular surfaces and margins, and presenting no trace of reparative action, the ulceration is said to be phagedenic. Many other varieties of ulcers are mentioned, but they all have reference to the amount and character of the exudative and reparative processes taking place; and though they afford excellent indications of the condition of the general system, which are well worth studying, they are not to be regarded as containing anything special in the nature of the ulceration itself. Rokitansky considers that the main circumstance determining ulceration, is the corrosive quality of the exudation, the ichor. We agree with Sir J. Paget, in doubting the correctness of this, as a general statement; it is much more probable that, in consequence of altered and defective nutrition, the tissue gradually deliquesces (so to speak) into a fluid, the cellular elements undergoing rapid fatty degeneration and decay. Commonly also the new products formed are themselves involved in the destructive process. Thus in tubercular and typhoid ulcerations of the intestines the process commences with the formation of a mass of new growth under the mucous surface. This causes absorption and destruction of the epithelium covering it, and thus an ulcer is formed, which is extended by the ejection of the newly formed mass.

It is a matter of some dispute whether the tissue, as it decays and is destroyed, is removed by absorption, or is cast off from the broken surface. Sir J. Paget inclines to the opinion that it is ejected, resting upon the analogy of excreting surfaces. on the discovery of fragments of bone and phosphate of lime in ulcers of osseous structures, and on direct observation of the commencement of ulcers. We are also inclined to think that the process of removal is rather by ejection than by absorption, especially in the case of open ulcers, yet so that some amount of absorption also takes place, varying in degree in different cases, and probably even predominating in those where there is no external outlet. The formation of ulcerations on the surface of the cervix uteri has appeared to us to take place in the following way, much as it is described by Dr. Baly on the intestinal surface:—As the first step, in the situation of a spot of hyperæmia, a minute vesicle is formed, the epithelial layer being lifted up by effused fluid, while the tissue beneath is softened, loosened up, and appears less dense than natural. Afterwards the covering of the vesicle is detached, the fluid escapes, and the tissue beneath appears still more lax and spongy, and has evidently undergone loss of substance. The hyperæmia persists. In this case we feel little doubt that the deliquescing tissue is partly thrown off in the fluid which escapes from the vesicle, partly absorbed by the blood-vessels.

It seems desirable to indicate the difference which exists between ulceration and absorption. In both there may be considerable loss of substance at some one or more points of the part affected, but in ulceration there is always an unhealthy state of the nutrition of the tissue, there is disease of it; in absorption this is not the case; the part may be diminished, but cannot be said to be diseased. Contrast a bone, carious and ulcerated from inflammation, with one which has undergone absorption, in consequence of the pressure of an aneurism.

Gangrene.—The last result of inflammation which we have to mention is Gangrene, or Mortification. This, indeed, is not a very common termination, nor is it at all peculiar to the inflammatory process. It more really belongs to a deficient condition of vital power induced by various causes, which may of itself be the cause of the death of some part, or render it so feeble that it perishes under injurious influences which would otherwise have had no such effect. Gangrene may ensue from the following causes: (1) from an absolute and prolonged stagnation of the blood; (2) from a defective supply of blood; (3) from a general taint or unhealthy crasis of the mass of the blood; (4) from a local injury. The absolute stagnation of the blood in the first case may be the result of violent inflammation, especially of an asthenic kind, and occurring in debilitated systems and organs; or it may be brought about mechanically, as when a portion of intestine is strangulated. Rokitansky says, that in this case the blood stagnant in the vessels first undergoes gangrenous decomposition, and that exuding through their walls in the state of gangrenous ichor, it sets up the same decomposing change in the surrounding tissues, which break up into a dark-coloured pulp, of as little consistence as tinder—diffluent, and excessively stinking. In the second case, besides various kinds of obstruction of the arteries from external pressure, their channels may be blocked up by extensive fibrinous coagula, either forming spontaneously or in consequence of disease of the coats of the vessels. They may again be plugged by blocks (*emboli*) transported from a distance. Gangrene occurring in aged persons, without any apparent cause, and that from the use of diseased grain and hospital gangrene, are instances in which the morbid action is dependent on a general taint of the blood, or decay of the whole system. Mr. Simon suggests that the mode in which ergot of rye produces its fatal effect may be by causing such contraction of the blood-vessels as prevents the flow of blood into the more distant parts, which consequently fall into the condition of dry gangrene.

Spontaneous gangrene in old persons, or others, in which after death no obstruction of the blood-vessels is found, can only depend on an actual and premature loss of vitality in the part affected, the tissues of which are no longer able to carry on the actions of vital chemistry, and yield to those of inorganic, *i.e.*, decompose, before the death of the system has actually occurred. In gangrene from local violence, or from frost-bite, &c., the vitality of the tissues of the part is destroyed by the injury done to them. The general characteristic of gangrene in all these cases is the failure of vital action; decay and death in the tissues, intense inflammation, absence of blood-supply, a poison circulating in its current, senile decrepitude, a fearful laceration, may all have the effect of dissolving the vital affinities which hold together the elements composing the complex substances of our organism, and allowing them to fall back, as they naturally do, into the simpler compounds of inorganic chemistry. Gangrenous parts which are exposed to the air are essentially putrescent, and contain both the usual chemical products of putrefactive fermentation and the organisms (Bacteria) which accompany or cause this process.

Varieties of Gangrene.—The distinctions of dry and moist gangrene, of black and white, of inflammatory and cold, have reference very much to the state of the affected part, with regard to the supply of blood. If the gangrene have its origin in inflammation, there will be a considerable quantity of fluid ichor effused, and the colour of the part will be of a deep red, or almost black. On the contrary, if the gangrene depend on deprivation of the supply of blood, the part will be more dry, and of a pale colour. Sometimes, especially from the effect of ergot of rye, a limb dries and shrinks up, becomes mummified, as it is said, with little change in colour. A black colour is, however, often observed in parts affected by *gangrena senilis;* this, no doubt, depends on alteration of the blood in the vessels, though there is often no hyperæmia. Soft tissues are more liable to mortify than such as are of a firmer consistence, bones, elastic and fibrous tissues resist longer than muscle and mucous membranes; the large vessels and nerves are sometimes seen completely exposed by the ravages of hospital gangrene, all the tissues being removed from around them.

The constitutional disturbance which often supervenes on gangrene is easily to be accounted for by the absorption of decomposing matters into the blood, which act as a virus upon it, and render it unfit to maintain healthy action.

PYÆMIA AND SEPTICÆMIA.

These conditions or diseases, as they are among the results, though only occasional ones, of inflammation, may very properly be considered here. The name Pyæmia was originally intended to mean (as it does etymologically) the presence of pus in the blood; and it was supposed that under certain circumstances, when suppuration occurred in one part of the body, pus made its way into the veins, and was thus distributed to various other parts, where it was *deposited;* hence the familiar surgical term "secondary deposits." It is not now believed that actual pus enters the veins; but in discussing the subject it is almost necessary to assume that some morbid poison, whether derived from inflammation or not, does enter the blood. We shall therefore endeavour to explain the morbid lesions on this hypothesis, and afterwards show what evidence can be given as to the existence and nature of this poison. The phenomena most frequently observed in pyæmia are somewhat as follows:—A man has received an injury, or undergone some surgical operation; it may be an amputation, or that for fistula *in ano;* for a time all proceeds well, but soon shiverings come on, with adynamic fever and oppression; he emaciates, pain or disorder shows itself in some internal viscus, and in a few days he dies in a state of stupor or delirium. Often the discharge of pus (if there have been any) from the original wound stops, granulations cease to grow, and it becomes what surgeons call unhealthy. The blood often contains an excess of leucocytes. On opening the body the blood is sometimes found abnormally fluid; but this is not constant: there are abscesses, more or less numerous, commonly in the lungs, and often in the liver, or other parts; there is frequently purulent inflammation of the joints or serous membranes, and often ecchymosis on the latter. Pyæmia may occur without any external wound, and then often assumes a chronic form. Many cases of "Puerperal Fever" also fall under the same head.

Experimental Evidence.—The original view of the origin of pyæmia was supposed to be confirmed by some of the earlier experiments. Cruveilhier, for instance, injected mercury into veins, and found that small abscesses were formed in the next set of capillaries supplied by the veins; *i.e.*, either in the lung, if the mercury was injected into veins of the general system; or in the liver, if into veins of the portal system. A small globule of mercury was found in the centre of each of these abscesses, showing that this had excited inflammation, passing into suppuration about itself in the part where it was deposited. The pus-globule was supposed to act in the same way as the globule of mercury; being too large to traverse the capillary channels it was arrested there; and similar obstructions taking place in other points of the same organ a number of separate inflammations were established, which suppurated and formed the so-called multiple abscesses. Since the time of Cruveilhier an enormous number of experiments have been made in the same direction, by injecting pus itself, as well as a number of other substances, neutral or irritating, into the blood or into the connective tissue. These experiments have led to conclusions which may be thus summarized:—(1) The secondary abscesses are true inflammations, not *deposits* of pus. (2) The starting point in their production is the obstruction of the capillaries by some substance which (like Cruveilhier's mercury globule) causes local inflammation. (3) This substance is transferred from the seat of primary inflammation to those of secondary inflammation. It is, however, quite certainly not pus.

Classification of Experimental Results.—The experiments which most concern us here may be arranged under three heads:—(1) Injection of pus into the veins or subcutaneous tissue; (2) injection of liquids containing septic or other supposed poisonous substances; (3) introduction into the veins of small solid masses or powders of various kinds. (1) Injections of pus produced very discordant results, till it was seen that the effects of liquid constituents and of any solid particles which the pus may contain are very different. Hence the later experimenters have carefully distinguished the effects of filtered pus or simple pus-serum from that of the unfiltered liquid. In the former case there is one constant symptom, viz., fever, or rise of the body temperature,* but metastatic abscesses do not occur, and perfect recovery may take place. The injection of pus containing solid masses or flocculi was found (especially by O. Weber) to cause, first, obstruction of the capillary circulation in parts of the lung (capillary embolisms), and finally the formation of abscesses in the parts thus affected. (2) The injection into the veins of various fluids derived from gangrenous inflammations, infusions of putrid matters, putrid pus, &c., if carefully filtered, has never caused the production of actual abscesses in the lungs, though it has caused local disturbances of circulation, ecchymoses (Savory), and various constitutional symptoms with which we are not now concerned. (3) Introducing small solid masses into the veins has the unavoidable result of causing obstructions of circulation (*i.e.*, arterial or capillary embolisms) with the results described in another part of this work,† and if the substances introduced are non-irritating (wax, india-rubber, &c.) nothing more. But if substances are used which mechanically wound and injure the vessels, as fragments of elder pith (Virchow) or charcoal, inflammation is produced, the point of obstruction sometimes leading to abscess. Chemically irritating substances, as mercury (Cruveilhier and Panum), produce still more marked inflammation, leading to abscess; and the most intense suppuration of this kind is produced by putrid or decomposing clots. Extremely fine powders produce capillary obstruction, but if they are non-irritating no further result ensues (Savory) than the ordinary results of capillary embolism elsewhere described (viz., infarction and hæmorrhage). It is clear then that the superaddition of inflammation to capillary obstruction depends not on the fineness of the obstructing substance, but on its nature, whether capable of exciting inflammation or not.

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* The evidence on this point is too clear and unanimous to be entirely invalidated by Stricker's discovery that the injection of *any* liquid, for instance water or even the animal's own blood into the veins, has some effect in causing a rise of temperature.

† See "Embolism," in chapter on "The Organs of Circulation."

Theory of Pyæmic Abscesses.—From the experimental results before stated has been deduced the theory of the origin of *Pyæmic abscesses*, now generally adopted, at least, as regards the lungs—viz., the *embolic* theory. It is this: that some solid matters capable of exciting inflammation pass from the veins of the part primarily inflamed into the pulmonary artery, and there produce arterial or capillary embolism, followed by inflammation and abscess. Similar, but not identical processes, lead to suppuration in other parts. The morbific material is believed to be disintegrated fibrin derived from the breaking down of clots in the veins of the affected part, that is, from *thrombosis*.

This is, however, not to be taken as a complete theory of pyæmia. It simply explains the occurrence of disease at particular parts of the body, and in a particular order, by the distribution of morbid material; but does not tell us what the disease is, or what is its *materies morbi*. We shall now apply this theory to the explanation of the most striking points in the morbid anatomy of pyæmia.

Characteristic Lesions.—The lesions which characterize pyæmia are, in Dr. Bristowe's words, "local congestions, extravasations of blood, inflammatory deposits, abscesses, and necroses." Congestion is a real and important link in the chain of pyæmic events; extravasations of blood are almost always found, either as petechial spots or as infiltrations in the substance of organs forming blocks or infarctions; inflammation is never absent, and true abscesses hardly ever; though some of what are called abscesses should be regarded as more strictly due to necrosis. The appearances in the lungs are the most important. Here we find (1) blocks which in their early stage much resemble hæmorrhagic infarctions, and are sometimes actually apoplectic; (2) sometimes merely inflammatory masses or blocks of lobular pneumonia. Both these seem liable to soften and break down, forming abscesses or cavities, containing puriform fluid; but the latter very often consisting of *débris* of tissue, oily particles, and disintegrating cell forms, without any true pus. The hæmorrhagic blocks may also be seen in various stages of involution, decolorized and softened, so as to form abscess-like cavities. The outer part of the block and some surrounding tissue is always, in both kinds of blocks, extremely congested and inflamed. This appearance of a yellow central portion, surrounded by a zone of intense hyperæmia, is very characteristic of pyæmic blocks; the pleura covering them is also always inflamed. The cavities thus formed may come to contain true pus, or true abscesses may occasionally be formed (as it would seem) by some more rapid process; but the occurrence of pus is far from universal, and can hardly be called very common. Another most important point is the condition of the pulmonary vessels. "The ultimate arterial twigs distributed to the masses of diseased lung structure seem to be invariably occupied, indeed distended by a soft pulpy yellowish material, or by something more like ordinary coagulum, consisting merely of disintegrated fibrine, with *débris* only of cells." Similar coagula have been found in small vessels leading to diseased patches in other organs. Sometimes a comparatively large arterial branch is found blocked with similar material; and a proportionately large mass of tissue in a state of inflammation or necrosis. With respect to the abscesses in parts other than the lungs, it must be admitted that their origin is not so easily explained on the embolic theory; but in most cases we can trace the three stages of vascular obstruction, extreme congestion or infarction, and necrosis or suppuration. The obstruction has, however, quite as much the appearance of arising from simple coagulation as from an embolic process.

Changes in the veins of the originally diseased parts.—Some of these are most often, though not always found inflamed, and with their walls indurated, and containing coagula, which resemble those just described in the pulmonary vessels, adherent to the walls, more or less decolorized, and usually in part at least softened down internally to a "reddish or yellowish puriform pulp or fluid," consisting generally of disintegrated fibrin. Dr. Bristowe has found both these and the corresponding arterial clots to contain in a few cases true pus, which is not surprising, though certainly exceptional. The phenomena are in fact those of thrombosis (which will be discussed in another part of this work), or local coagulation of blood, followed in this case by rapid disintegration of the clot, and accompanied by inflammation of the walls of the vein.

Comparing the details of morbid anatomy, just given, with the theory before enunciated, it is plain that they correspond very closely, and that, so far as pyæmic abscesses, especially in the lungs, are concerned, the *embolic* theory is a sufficient explanation of their occurrence. Softening of the vein clots causes disintegrated fibrin to pass into the circulation, which then obstructs the pulmonary circulation, and excites inflammation there. Certain difficulties, however, have been raised as to the occurrence of abscesses in other parts than the lungs, when the latter are free. How can the particles which cause suppuration pass through the lungs, and cause capillary obstruction and its consequences in other parts, as for instance, the liver? O. Weber attempted to answer this question by experiment, and found that as a matter of fact he could inject into the veins flocculent pus, so fine that it passed through the pulmonary capillaries, and was arrested in the systemic circulation, where (*e.g.*, in the liver) it produced capillary embolism, proved by microscopic examination, and abscess. When there is coagulation in the pulmonary capillaries it is also known that this may extend into the pulmonary veins, and thus a second set of thrombi be produced. The other points in the morbid anatomy of pyæmia also admit of explanation to some extent. Hæmorrhage, or ecchymosis, is a direct consequence of capillary obstruction, when the embolic material is of such a nature as to injure the vascular wall. Thus Feltz, in experiments on living animals, saw a hæmorrhagic patch actually forming, in consequence of the obstruction and injury of capillaries by injected substances.

Little has been done to explain the origin of inflammations in joints and serous membranes.

Properties of the Venous Clots.—Supposing that the matter of decomposing clots does pass into the circulation, and produce the results just described, we still have to inquire how the clots come to break down, and how they have infective properties, which certainly do not belong to all crumbling fibrin. It has been found, as a matter of fact, that the coagula are readily formed, and do readily break down in veins which are surrounded by tissues in a state of inflammation; but especially when this is an unhealthy or "septic" inflammation. These results have been attributed to inflammation of the vein, or to the infiltration through its walls of some poisonous substance derived from the inflamed parts, or even to the passage of leucocytes through the walls. At all events the softening is not very different from that which occurs in other situations (for instance, in the heart), though it appears to be more rapid. The infective properties of the clots are, however, a matter of demonstration; and it must be supposed that they carry with them the unknown poison generated in or introduced into the wound.

Objections to the Embolic Theory.—It is not to be denied that these phenomena in veins are often absent; and some observers have gone so far as to say more often absent than present. But we must point out that the small veins are not always carefully examined, and yet such a vein is extremely likely to be the starting-point of the disease. Again, many cases of pyæmia start from bone, and veins in bone are hard to trace. The occurrence of some morbid process in the veins is rendered very probable, by the fact that in cases of pyæmia, without external wound, there is often coagulation in some veins, and subsequent breaking down of the coagulum.*

Those who reject the embolic theory, or find it insufficient, have supposed that the blood acquires from the entrance into it of some poisonous matter derived from inflamed parts the property of coagulating very readily, and that this property explains the obstruction of the minute vessels and capillaries by clots. If this were so the coagula ought, one would think, to be more general than they are. Again, it has been supposed that the corpuscles of the blood acquire some property which may be termed adhesiveness, which causes them to agglomerate together, and to block up the minute capillaries. But obstruction of capillaries by such masses has not been observed in pyæmia, though it has been in other diseases in a few instances. Whichever of these views be adopted it is clear that the next important question is whether any morbid poison does enter the blood from the inflamed part; and if so, what this poison is. We therefore return to examine the hypothesis which we assumed on setting out.

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* There are doubtless other changes which may replace the softening of vein-clots as the primary disease: *e.g.*, osteo-myelitis. So common is this in pyæmia from bone injuries, that some military surgeons have spoken of the two diseases as almost convertible terms. Now, the red osseous medulla is believed to be one of the parts which furnish leucocytes to the blood, and has other relations to the veins which particularly fit it for pouring morbid material into the circulation.

Specific Theory of Pyæmia.—The embolic theory, as well as others, requires the assumption that some morbid poison is distributed through the body from the primary seat of disease; but opinions differ greatly as to the probable nature of this poison. Some have attempted to discover it by chemical means, and have extracted from the putrid matters supposed to excite pyæmia a number of substances, such as ammonia, sulphuretted hydrogen, sulphide of ammonium, and various products of decomposition, which, when introduced into the blood of animals, have produced various morbid symptoms, but not pyæmia. Decomposing albuminous matters have also been supposed to act as a ferment on the blood. More recently a substance, to which the name of *sepsin* has been given, has been isolated, in the form of a combination with sulphuric acid, and even crystallized. It is said to produce all the poisonous effects of putrid substances in a most intense degree. It appears to be soluble in water, not volatile, not destroyed by boiling or evaporation to dryness. But it must remain for the present doubtful whether this is a pure substance.

On the other hand, there are many who regard the hypothetical poison as an organism of the group Vibriones or Bacteria. Recklinghausen has observed such forms, which he calls micrococci, in pyæmic abscesses. Burdon Sanderson has traced similar organisms in the fluids of a large class of infective inflammations. Klebs believes it to be a true fungus, which he calls *microsporon septicum*, and several other pathologists have made similar observations. It seems to be clear, however, that the ordinary bacteria found in putrefying fluids cannot produce pyæmia; though they may, if injected into the blood, produce marked constitutional derangement, and also obstruction of capillaries. This subject is further discussed in the section on Vegetable Parasites.

On the whole, we must conclude that pyæmia is a general disease produced by some morbid poison, either self-generated or introduced from without; but that the nature of this poison is not yet known. The most prominent lesions (abscesses and blocks) found in many cases of pyæmia, are not essential to the disease, but the result of secondary processes, which effect a peculiar distribution of the poisonous substance. Cases of pyæmia in which such lesions are absent are nevertheless extremely difficult to distinguish from the other conditions into which they pass by insensible gradations, viz., fever resulting from suppuration or surgical fever (called by some writers pyæmia simplex), and the disease believed to be due to the presence in the blood of putrid or septic material, called septicæmia. The former does not come within the scope of this work; of the latter we must give some account, though leaving it undetermined whether it is really distinct from the constitutional condition of pyæmia. The two diseases appear to be often combined.

SEPTICÆMIA.

This has been also called ichorrhæmia, septic pyæmia, or putrid infection. It occurs rarely (some would say never) as an uncomplicated affection. When it supervenes after an injury the following symptoms are observed. At an early date, usually two or three days after the injury, the wounded part (which may never suppurate) assumes a swollen, infiltrated, and discoloured aspect, or inflammatory œdema, which may become actual gangrene. The febrile action runs high at the commencement, but soon subsides, and the temperature may even fall below the normal. Rigors rarely occur at the outset, and recurring shiverings do not appear in the course of the affection. The urine sometimes contains albumen. The skin is relaxed and dry; though in rare instances profuse perspiration may usher in the attack. Delirium is not constant, but apathy, passing into stupor, is usually observed. Copious diarrhœa sometimes occurs. The disease runs a very rapid course, terminating fatally even in a couple of days—but in some instances lasts six or eight days, or even more rarely two or three weeks.*

Morbid Anatomy.—This is not at all characteristic; the blood is usually dark coloured, and imperfectly coagulated, the spleen enlarged, and soft; liver and kidneys sometimes enlarged. The intestines occasionally exhibit hæmorrhagic patches and swelling of the follicles, and contain black liquid fæces. Multiple abscesses do not occur; but in the experience of military surgeons dark coloured metastatic nodules may occur in the pulmonary tissue, which, instead of undergoing suppuration, become gangrenous. Very rapid post-mortem decomposition is the rule.

The above symptoms and post-mortem appearances correspond pretty closely with those observed in animals after the experimental injection of putrid animal matter into their veins. But it must be said that the type of disease presented by such animals is more definite and recognizable than any derived from observation of actual cases of disease in man. And if we call the experimental disease septicæmia, the clinical disease seldom perfectly resembles it. Our own experience has supplied us with a few cases which appear to fall under this head. They are those in which death has followed some injury complicated with gangrene, bed-sore, urinous infiltration, what has been called pseudo-erysipelas, &c. The most remarkable post-mortem phenomenon is the extremely rapid decomposition of the body, and the enormous evolution of gas; so that even in winter, and less than twenty-four hours after death it may be swollen and discoloured in as frightful a manner as in summer after some days. Numerous cavities containing gas are found in the viscera; and on examination both these and the blood are found to swarm with the bacteria of putrefaction. The occurrence of these at such a time after death, and in such numbers, is so exceptional that it must be regarded as the consequence of disease existing during life; and considering that the diseased parts (gangrenous wounds, foul bed-sores, parts infiltrated with urine) also swarm with bacteria during life, it seems reasonable to conclude that these bacteria were distributed by the circulating blood to various parts of the body; though the most conspicuous results of their activity are manifested after death. Of course the chemical substances which always accompany bacteria in putrefaction must also have been present. In this sense we understand the word septicæmia. Since, however, both here and in the corresponding experimental disease, chemical products as well as bacteria are present, we cannot tell which should be regarded as the morbid poison. In any case the bacteria must not be confounded with the specific organisms which are supposed to be the cause of pyæmia or other specific diseases; since these, if they exist, are certainly distinct from the ordinary putrefactive forms (Cohn).

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* Fitzgerald, Appendix to Report of Army Medical Department for 1871.

The following more recent works may be referred to on pyæmia and septicæmia:—

Virchow. "Gesammelte Abhandlungen." 1856.

Bristowe. Article Pyæmia, in "Reynolds' System of Medicine." Vol. I. 1866.

Hueter, in Pitha und Billroth's "Handbuch der Chirurgie." Band I. Abth. 2. 1869.

Savory. "St. Bartholomew's Hospital Reports," 1865, 1866, 1867.

O. Weber. "Deutsche Klinik." 1863, 1864, 1865. *s.v.*

Klebs. Abstract from a Swiss Journal, in Virchow's "Jahres Bericht" for 1870. Vol. I.

Billroth. "Surgical Pathology" and Papers in "Archiv. für Klin. Chirurgie," 1862 to 1867.

Feltz. "Des Embolies Capillaires." 2nd edition. Paris. 1870.

Burdon Sanderson. "Pathological Transactions," vol. xxiii., p. 303, 1872; "Medico-Chirurgical Transactions," 1873; "Reports of the Medical Officer of the Privy Council."

Cohn, on Bacteria; "Beiträge zur Biologie der Pflanzen," Part II., 1872; "Quart. Journ. Micr. Science," April, 1873.

CHAPTER IV.

TEXTURAL CHANGES.

We now come to consider certain changes, to which most of the various organs of the body are liable, in a general way. These changes are essentially textural, and result from various disturbances of the normal degree and kind of nutrition. They are also for the most part slow and gradual in their course, and are thus termed chronic.

Hypertrophy, as its etymology signifies, conveys the idea of increased nutrition and growth in the part affected. The term, however, is sometimes applied to parts which are simply enlarged, and it is essential to observe that this enlargement by no means necessarily constitutes hypertrophy, but may, instead, be attended with the opposite condition. This makes it necessary to distinguish *real* from *apparent* hypertrophy. In the former the characteristic tissue of the part is enlarged, and more developed: if it be a muscle, the muscular fibres grow larger, and attain to greater energy of contraction; if it be a kidney, more renal tubes are formed with corresponding blood-vessels. The size of the organ is not only increased, but its working power too, the muscle can raise a greater weight, and the kidney can produce more secretion. But if a liver or spleen be enlarged by ever so great a quantity of the peculiar matter termed "waxy," which is deposited interstitially between the elements of the tissue, their functional power is only thereby lessened and deteriorated, for the simple reason that the new substance has pressed upon and caused wasting of the natural structure. It is therefore necessary in every case to ascertain what is the nature of the enlargement of a part before we pronounce it to be truly hypertrophied. Mere distension of a hollow organ, of course, is not hypertrophy; a huge emphysematous lung, or hydrocephalic brain are not really enlarged, but rather diminished in actual capacity. Great congestion of a part with blood may give it the appearance of being hypertrophied, but this again is only another kind of distension. Real hypertrophy requires a free supply of healthy blood, and is commonly attended with enlargement of the vessels of the part; this is not the case in apparent. The cause of real hypertrophy in active organs seems to be always the increased exertion of the organ, more than usual effort is demanded of it, and according to the law of the circulation which we have noticed, more blood flows to the part than usual; this, if the organ be in a healthy state, not only supplies its waste, but furnishes material for increase and development. The heart in various diseased states of its valves, the urinary bladder in stricture of the urethra, the remaining kidney when one has been destroyed, the muscles, and even the solid bones themselves, when long and actively exercised, afford excellent examples of true hypertrophy; which may in these cases be called *functional.* There may, however, be simply nutritive or passive hypertrophy; like that of the cock's spur which John Hunter transplanted from its foot to its head, and which there grew to a very large size. Examples of this are seen in the rare cases where one side of the body or one limb grows to a gigantic size; or is simply larger than the other. Again, conditions which cause an increased flow of blood to the part may sometimes (though not always) give rise to hypertrophy. Inflammation may have hypertrophy for its permanent consequence, as in enlargement of bones from chronic periostitis.

Functional hypertrophy, though in several instances it brings about an abnormal state of the part, is yet for the most part not to be considered in the light of a disease. It is really a compensatory effort made by the system to obviate as far as possible the evils that arise from some damage that an important part has sustained. Thus, if we find the walls of the heart greatly thickened, and its power proportionately increased, we should naturally fear that such an abnormal increase of power would prove a cause of danger to the system, and would probably induce hæmorrhage in the brain, or elsewhere;—but if we know that at the same time there exists regurgitant disease of the mitral or of the aortic valves, then we see that the hypertrophy, so far from being attended with danger, is useful and necessary to enable the circulation to be carried on against such impediments.

Hypertrophy usually depends on multiplication of the elements of the part, not on enlargement of them; the ultimate fibrillæ of muscle in a hypertrophied heart for instance are of the normal size. This multiplication has been called *hyperplasia;* but such condition does not always lead to hypertrophy. It only means that new elements like the old ones are produced; but these new elements may quickly perish as they usually do in inflammatory hyperplasia; or they may be changed into something different from the original elements as in new growths, many of which begin with a process of hyperplasia.

Atrophy is the opposite condition to hypertrophy; and is commonly conceived of as implying a wasting and diminution of the part. Atrophy, however, may have taken place to a great extent, without any diminution, but an increase of size. These are the instances of false hypertrophy, to which we have above alluded.

In a few cases atrophy is a natural process, as in the disappearance of the thymus gland when the age of early infancy is passed. Inactivity of a part, obstruction of its blood-vessels, failure of its own vital energy, continued pressure upon its surface, are all recognized causes of atrophy. A muscle, if unused, becomes small and pale, and its tissue degenerates; the bones of a paralytic limb lose in density and strength, and in compactness of tissue; the brain in second childhood shrinks within its bony case, and leaves a space occupied by serum. Obstruction of the arterial branch leading to a part of the kidney will cause wasting of the epithelium of the tube in that part, ligature of the thyroideal arteries has caused considerable diminution of a goîtrous tumour. Thinning of the walls of the heart, renal degeneration, the fall of the hair, and the general decay of advanced age, are instances of atrophy from failing vital energy of the tissues. With the effect of pressure in producing atrophy, all are familiar; it is well exemplified in the absorption that takes place from the pressure of aneurism, which affects not only the soft parts, but the bones themselves. In most cases, atrophy is an actually morbid process, and is attended by a change in the condition of the elementary parts of the tissue which attests the unhealthy character of their nutrition, especially by some of the forms of degeneration mentioned below. Atrophy often occurs as a secondary process, induced by some primary one, which may have been attended with apparent hypertrophy. Of this the liver in the earlier and later stages of cirrhosis furnishes an instance.

Induration and *softening* are terms that are commonly employed to express changes that have occurred in the consistence of various organs, rendering them more or less firm and dense than natural. They are, of course, very general in their meaning, and of themselves tell nothing as to the pathological condition of the part affected. This must depend entirely on the causes of the changes in question. The commonest cause of induration is the effusion of fibrinous material into the interstices of a tissue; if this does not liquefy and become absorbed, it passes into the state of fibroid tissue, and being blended with or replacing the elements of the part, it occasions a more or less considerable increase of density and toughness. Instances of this are extremely frequent in the lungs around tuberculous deposits, in the cirrhotic liver, and in the areolar tissue around ulcers. Induration may also be caused by mere multiplication of the elementary fibres or even simple thickening of them; as is seen in induration of the kidneys from cardiac obstruction. The tissues involved in the induration matter, as it is often called, are very apt to become atrophied, partly in consequence of their supply of blood being cut off by obliteration of the vessels distributed to them, partly from the effect of simple pressure. According to its seat, induration may be of trifling consequence, or very serious; in the general areolar tissue of the body it may only cause slight impediment to the free movements of a part; in the valves of the heart it is a common cause of secondary disease, dropsy, and death. Textures are often rendered harder and firmer by other deposits than simple fibrinous, as by tuberculous, bacony, calcareous, but to these the term induration is not so strictly applicable.

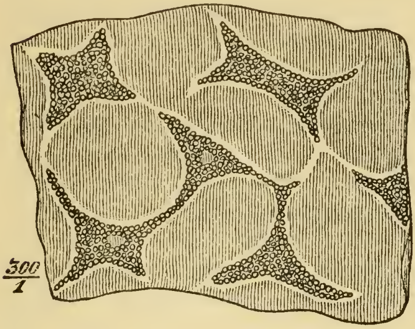
Softening of a part may be brought about by very various causes. It is almost an invariable effect of acute inflammation actually existing; it is also found as the result of inflammation that has in great measure subsided; it occurs from deprivation of blood, as a kind of atrophy, and probably, in some cases, as a local result of a general cachexia. In all cases it involves a considerable deviation from the state of healthy nutrition, and if it proceeds far, may easily occasion a breaking down and destruction of the tissue. The distinction of various kinds of softening, especially of the red, or inflammatory, has often been considered difficult, but may generally be made with certainty by means of microscopical examination, which discovers in the former decided traces of exudation. Softening is connected with the hypnotic condition of the blood, and allied states, especially the typhous; induration, on the other hand, with the fibrinous crasis. Softening is more prone to occasion speedy destruction of textures and fatal disorder. Induration, to produce gradual changes whose effects are slowly and gradually manifested. Softening appears as a process of decay, and affects not only natural structures, but new formations, and even as we have seen tuberculous deposits and fibrinous coagula. Induration, on the contrary, though involving some degree of atrophy, tends to preserve the parts which it affects from entire dissolution.

DEGENERATIONS.

Degenerations are changes of an essentially chronic nature, latent in their origin, and obscure in their progress, until they have produced such deteriorations of structure as give rise to prominent secondary phenomena. Those with which we are most acquainted are the fatty, fibrous, calcareous, and waxy or amyloid. They are, except the last, of extremely frequent occurrence, but their nature has scarcely been recognized until of late.

Fatty degeneration consists in the replacement of the healthy tissue of a part, by drops, or molecules of oily nature, which are deposited, as it seems, instead of the natural material. This is the case with the substance of the connective-tissue corpuscles shown in the figure (p. 124). This character distinguishes it from fatty accumulation or *infiltration*, which may take place to a great extent in the interstices of a tissue so as to overlay and conceal its elements. In true fatty degeneration there is always destruction of tissue, which does not occur when there is merely an increase of oil in the substance of the part. A muscular fibre thus affected shows the sarcous elements, the real contractile tissue within the sarcolemma, replaced by glistening oil particles, so that the functional power of the organ is *pro tanto* destroyed. The hepatic cells in true fatty degeneration not only fill themselves with oil, but fuse together with others, and break up into granulous films, entangling oil-drops; this destruction does not occur when they simply become loaded with oil from the presence of a large quantity of this substance in the food. The process by which fibrinous coagula, or extra-vascular deposits are broken down and dissolved, seems to be in some measure of the nature of fatty degeneration; there is commonly much free oily matter visible in the softened mass, and the exudation corpuscles seem to be thoroughly charged with it. Fatty degeneration is clearly a kind of atrophy, but not identical with the simple form; we have seen muscular fibres of the heart which were simply atrophied, and had lost their transverse striation entirely, which yet did not contain a single particle of oil. The prevalent opinion respecting the nature of fatty degeneration is, that there occurs a true conversion of the albuminous substance of the tissue into fatty matter, just as when adipocere is formed out of flesh immersed in water, but this is opposed by Dr. Ormerod's investigations. However this may be, it is important to distinguish the following conditions in which the quantity of fatty matter in and upon a part is greatly increased. (1) Mere accumulation of adipose substance in and around an organ, the tissue remaining healthy; (2) accumulation of adipose tissue in the same way, but with atrophy of the proper structure; (3) increase of oil in the elementary structure of a part without atrophy, or breaking up; (4) true fatty degeneration, in which the structure is more or less destroyed, and its elementary parts converted into oily matter.

FIG. 14.



Connective-tissue corpuscles from the inner coat of an artery, in a state of fatty degeneration. (After Rindfleisch.)

It is remarkable that poisoning with phosphorus produces marked fatty degeneration of several elementary tissues; as muscle, liver cells, and other glandular cells. In this case it seems to have been proved that the fat is formed from the albuminous matter of the tissues, some nitrogenous substance, which ultimately decomposes into urea, being formed at the same time.

Fibroid degeneration is somewhat allied to induration. It occasions the gradual thickening of serous membranes and of areolar tissue by the formation of an imperfect kind of fibrous structure. This may attain a considerable thickness, and then by its dead white aspect resemble very much a layer of cartilage. The capsule of the spleen is sometimes thus altered, and has been wrongly said to have undergone cartilaginification, for there is no real similarity between this substance and cartilage. The white patches formed on the surface of the pericardium and in the capsule of the liver are produced in this manner, and so is also that thickening of the Glissonian sheaths, which give rise, in many cases, to cirrhosis. Many organs show similar changes. Thus in the heart we may find the muscular tissue replaced to a great extent by newly-formed fibrous (or so called "fibroid") tissue. Large tracts of the lung may be converted into similar tissue in the disease called Fibroid Phthisis. Granular disease of the kidney ultimately results in a similar substitution. Since in all these cases the specialized tissue of the organs becomes replaced by one of less perfect organization, the change may be described as a degeneration. It has also been proposed to term it fibroid substitution. The new tissue is probably formed, in part, by thickening of the pre-existent fibrous tissue which exists more or less in all organs, in part also by nuclei, developing fibres by a process similar to that which takes place in the formation of normal connective tissue, or of that produced for repair. The chief difference between induration and fibrous degeneration consists in this, that in the former, a notable quantity of blastema is effused, which becomes the induration matter, and compresses and atrophies the adjacent texture; in the latter there seems to be scarce any perceptible exudation, as it takes place slowly, and passes at once into the condition of fibre. Induration may affect any tissue, while fibrous degeneration is chiefly seen in membranes. Cartilage, however, is liable to a fibrous transformation of a somewhat different kind, which will be hereafter noticed. Fibrous degeneration seems to be sometimes a consequence of long-continued venous congestion, as seen in the liver in cases of heart disease; and possibly of deficient supply of arterial blood.

Calcareous degeneration rarely occurs as a primary change, it is almost always secondary to some other. Especially it seems to be consequent upon a fatty degeneration of the arteries, to which the term *atheroma* is applied, and which may occur at any period of life, while the calcareous change is seldom observed very extensively, except in advanced age. It is often spoken of as ossification, and, indeed, not altogether without reason, as the "lacunæ" characteristic of bone are found in this substance also in some cases;—for instance, according to Virchow, in the calcareous plates of arteries, where they are, however, irregularly and imperfectly developed. The earthy matters deposited are, principally, phosphate of lime and magnesia, and carbonate of lime; Rokitansky considers them to be not so much new deposits as precipitations from their natural combinations with animal matters. Calcareous deposition seems generally to take place in parts whose vitality has been considerably lowered by previous morbid processes within them. Thus it is common in lymphatic glands which have been the seat of scrofulous disease, in obsolete croupo-fibrinous deposits, in the coats of arteries which have begun to be affected by atheroma, and in the valves of the heart under similar circumstances. The atheromatous condition, which we shall describe more particularly when we speak of the diseases of arteries, may either terminate in softening and breaking down of the arterial coats, or in calcareous deposition; both of these changes often co-exist, but the latter predominates in old age. We think, however, that deposition of earthy matter may take place to a great extent, so as to produce the ossification so common in the vessels of the aged, without previous atheromatous or fatty degeneration. The quantity of earthy matter in the bones becomes greatly increased in later life, it is even deposited in the so-named permanent cartilages, and it is, therefore, not surprising that it should also affect the walls of the vessels. This degeneration, probably, is occasioned solely by a failure of assimilative nutritive power in the tissue itself.

Other forms of degeneration have been described by authors; as the *colloid*, which affects the cells of certain tumours, causing them to lose their distinctness; becoming first individually converted into masses of transparent gelatinous material, and next fused together so as to lose wholly or partly their separate outlines. The material called colloid is related to the albuminates, and resembles mucin, but differs in not being precipitated by acetic acid. Colloid metamorphosis, or degeneration, occurs in normal structures, as the thyroid, and in new growths, as in "colloid cancer."

Mucoid degeneration, better called simply metamorphosis, consists in the transformation of albuminous constituents of tissues into *mucin*, a substance distinguished from albumen by the property of being coagulated by acetic acid, and not re-dissolved by an excess. This substance is often found in embryonic tissues, and the transformation in question occurs in tumours chiefly of the connective-tissue class, as fibroma, sarcoma, enchondroma; also in some normal tissues, as cartilage.

Pigmentary degeneration hardly deserves the name of a general pathological process. The darkening of many parts of the body by the deposition in them of black pigment is a phenomenon which often accompanies other changes, and is chiefly found in conditions of chronic hyperæmia or inflammation. Black pigment is doubtless derived from a chemical transformation of the normal colouring matter of the blood, which is either set free in the tissues by actual hæmorrhage, or merely transudes in consequence of stagnation. Certain parts of the tissues seem in general to have a peculiar power of absorbing the pigment; as is seen for instance in some connective-tissue cells of the membranes of the brain, &c. (see Fig. 31). The pathological pigmentation is often but an exaggeration of the normal, as in the skin, cerebral membranes, &c. This is true even of extreme cases of pigmentation, such as that in Addison's disease, where the naturally pigmented parts of the body become darker. Our figure represents the deposition of pigment in this disease in the connective-tissue corpuscles of the pia mater covering the medulla oblongata, which is often found specially dark.

Most lung pigment has a different origin, being carbon taken in with the breath. That in the bronchial glands is doubtless the same substance, conveyed from the lung by the lymphatics.

Waxy, Lardaceous, or Amyloid Degeneration.—This name is used to denote a degeneration which may affect almost any organ of the body, but is most common in certain of the internal viscera. The organs affected by it have, when the degeneration is far advanced, a peculiar appearance, which is, even to the naked eye, quite distinctive of the disease. They are pale, more homogeneous than is natural, somewhat translucent, and appearing generally as if soaked with some semi-transparent material. On section they are found to be firm and unusually dry. This change is not, however, always so universal, but may affect only special parts of particular organs. The change is found to be essentially due to the deposition in the affected structure of a peculiar material, the nature of which has been the subject of much discussion. Formerly supposed from its reaction with iodine to be allied to starch, and at another time wrongly identified with cholesterine, it has now been clearly shown to be a nitrogenous compound, of complex constitution, belonging to the large group of albuminates. Its precise composition has not been determined, in consequence of the great difficulty of obtaining it perfectly pure. It seems, however, to have a more complicated constitution than most albuminates. The waxy substance is not altered by the action of water, and shows a remarkable resistance to ordinary putrefactive change. It is also not dissolved by digestion with pepsin, as are other albuminous substances; and this property has been made use of for its isolation. Its most remarkable chemical reaction is the change of colour produced by the action of iodine. Parts which have undergone waxy degeneration are stained by tincture or watery solution of iodine (with potassium iodide) of a yellowish-brown, or sometimes a darker mahogany-brown colour. This change is usually seen in some of the elementary parts only, as in the smaller arteries, the glomeruli of the kidney, or sometimes in epithelial or glandular cells. More rarely there is a general colouring, or what is most usual, a general yellow tint is produced along with a darker colour of those parts more specially affected. These colours are distinguished from what iodine produces in healthy organs, both by their greater intensity and by their greater permanence. If the parts thus stained be further treated with dilute sulphuric acid, the yellowish-brown colour usually becomes nearly black, purplish, or in rare cases actually blue.*

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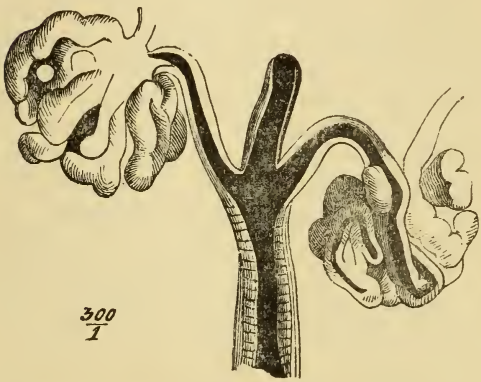
* In order to obtain a blue or purple colour in the iodine-stained structures by the action of sulphuric acid, the best plan is first to soak the specimen (well freed from blood) in dilute solution of iodine; then to place it under water, which covers it to the depth of half or a quarter of an inch; and finally to drop concentrated sulphuric acid from a little height upon the surface of the water above the specimen. In this way a

It is not certain whether the waxy or amyloid substance results from an actual metamorphosis of the substance of the tissue elements, or whether it merely infiltrates them, being poured out by the vessels. The latter supposition is supported by the fact that the change primarily affects, in most organs, the capillaries and smaller arteries. These structures are found greatly enlarged, the walls thickened, but the calibre much diminished, and presenting a peculiar pale, homogeneous, translucent appearance, all the minute structural characters of the part, the nuclei, fibres and cells being quite obliterated. In arteries it is thought that the change commences in the inner coat. Next in order of frequency to be affected by this form of degeneration are glandular cells; and in some organs, as the liver, these, not the vessels, seem to be primarily altered. Cells which have undergone lardaceous degeneration are enlarged, rounded in their outlines, homogeneous, translucent, and highly refracting; often two or more may be found fused together into an irregular mass. The anæmic condition of lardaceous organs is thus explained both by the impermeability of the vessels and by the swelling of the tissue elements, which, pressing on the vessels, hinder the supply of blood.

Distribution of Lardaceous Degeneration.—The organs most frequently affected are the spleen, liver, kidneys, and lymphatic glands. Second in order of frequency must (according to Vagner) be placed the mucous membrane of the intestinal canal, the suprarenal capsules, and the omentum. More rarely (and seldom or never without simultaneous affection of some of those organs just mentioned) the same change is found in the mucous membrane of other parts of the digestive tract, the pancreas, the mucous membranes of the urinary organs, the generative organs, the heart, thyroid body, lungs, and bronchi; as also, it is said, in blood clots.

Fig. 15.



Malpighian tufts, and a small artery of the kidney affected with lardaceous degeneration. (After Rindfleisch.)

Causes of Lardaceous Degeneration.—This affection almost always occurs as the sequel of some constitutional disease or exhausting

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powerful but momentary action of the acid results, which generally (though it sometimes fails) produces a permanent black or purplish colour in those parts previously stained by iodine.

local affection; very rarely it appears to be idiopathic, or at least the primary disease cannot be traced. By far the most frequent determining cause is some long-continued suppuration, such as accompanies necrosis or caries of bone, chronic abscess, joint disease, &c. In the same way it may follow pulmonary phthisis where there is much inflammatory destruction of the lung, dilatation of the bronchi with suppuration, empyema, chronic intestinal ulceration, pyelitis, cystitis, and, it is said, chronic ulceration of the skin. Syphilis is a very frequent precursor of waxy degeneration, and this usually when it takes the form of bone disease or chronic ulceration; but by no means invariably, for cases occur in which waxy degeneration of the kidneys is the most prominent symptom of constitutional syphilis. It is also believed by Dr. Dickinson that this degeneration of the kidneys may occur as a sequel to simple chronic inflammation of those organs (tubular nephritis), but the question will then suggest itself whether the waxy degeneration may not have been the primary disease.

A chemical explanation has been proposed by Dr. Dickinson of the fact that in the immense majority of instances this form of degeneration is preceded by chronic suppuration. He finds that in tissues affected with waxy degeneration the proportion of alkaline salts (as determined in the ash left by ignition) is remarkably diminished, even to the extent of 25 per cent.; while the proportion of lime is in some degree increased. He has also pointed out that parts thus affected have lost the natural faint alkaline reaction of healthy tissues, and are neutral or acid, so as no longer to discharge the colour of indigo solution, but to become stained by it. They may thus be recognized as clearly as by the iodine reaction. Regarding the lardaceous substance, in accordance with the latest analysis as allied to fibrin, Dr. Dickinson goes a step farther, and concludes that it is nothing more than fibrin deprived of the alkaline salts which it naturally contains. Hence he denominates it *dealkalized fibrin*, and has, by treating fibrin with acids, produced a substance which somewhat resembles the lardaceous material in its reaction with iodine. Finally, the loss of alkaline salts is attributed by Dr. Dickinson to the chronic purulent discharge, which is the usual antecedent of this disease, pus being found to contain a large proportion of potassium and sodium. The name of *depurative* disease is hence applied to this form of degeneration.

Space does not permit any full discussion of this hypothesis. We can only say that Dr. Dickinson's theory appears by no means satisfactorily proved. We do not know how far the deficiency of alkaline salts is peculiar to the state of waxy degeneration, or whether it does not occur in other anæmic conditions of the tissues. There is no obvious connection between the loss of alkaline salts (which may be neutral) and the change of reaction; and finally no proof has been offered that the fibrin, or any constituent of the blood, has actually undergone such changes as, according to the theory, it should have undergone.*

Consequences and Symptoms of Lardaceous Degeneration.—The normal functions of the organs affected are, generally speaking, suspended or seriously interfered with. In the case of the kidneys there is deficient excretion of urinary substances (though there may be superabundance of water), albuminuria, and dropsy. When the spleen, liver, and lymphatic glands are affected, there will be deficient blood-formation leading to anæmia and general cachexia. The intestinal affections give rise to copious watery diarrhœa. General dropsy is a common symptom. Purpura has been traced to this condition in some cases with much probability, hæmorrhage being permitted by the diseased state of the vascular walls. The general consequences and symptoms are often with difficulty to be distinguished from those of the previously existing constitutional state which produced the disease, and hence diagnosis is almost confined to recognizing the affections of particular organs; but only in the case of the kidneys, liver, and spleen is this possible. Waxy degeneration of the kidneys is made evident by a train of symptoms to be hereafter described, which are still by no means free from ambiguity. The same alteration of the liver and spleen may be concluded from enlargement, which cannot be traced to any other of the known causes of hypertrophy, especially if there have been a history of suppuration. The simultaneous occurrence of these symptoms in distinct organs of course increases the probability of the diagnosis in a very high ratio. Grave and inveterate cases of this affection show no tendency to recovery, and appear to be inevitably fatal. Nevertheless it is extremely probable that slighter forms of the disease, which escape notice, occur and pass away again; while some good observers assert that during the course of a chronic suppuration enlargement of the liver and spleen may be sometimes distinctly made out, and completely disappear when the primary disease gets well. In such cases recovery clearly depends upon removal of the determining cause.

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* A committee of the Pathological Society appointed to investigate this subject found that livers affected with lardaceous degeneration were deficient (as compared with healthy organs) in potassium and phosphoric acid, while they give an excess of sodium and chlorine; the proportions per cent. being as follows :—

	Potassa	Soda.	Phosphoric Acid.	Chlorine
Average of three healthy livers	·2821	·0948	·3295	·1073
Average of three lardaceous livers	·1319	·1902	·1981	·1664

They also found in one specimen of lardaceous liver a larger proportion of cholesterin than in the healthy organ, and generally an increased proportion of fat. Experiments made to ascertain the relation of the lardaceous substance to fibrin did not lead to any definite result. ("Trans. Path. Soc.," vol. xxii. p. 1, 1871.)

CORPORA AMYLACEA.

By this name are distinguished certain bodies which were at one time believed to consist of starch, and were both on that ground, and later on other grounds put under the same head as the material found in waxy degeneration. They are minute, mostly of microscopical size, though sometimes just visible to the naked eye; usually oval or round, and of concentric laminated structure, of unknown chemical composition, but coloured, either bluish or brown, by iodine. They are found in various organs, and it is not certain that those from different parts of the body are all of the same nature.

The corpora amylacea of the brain and nervous system form the most clearly defined and recognizable group. In all atrophic conditions of nervous tissue, especially in softening of the nerve-centres, the microscope shows a number of minute bodies not unlike starch granules, generally made up of concentric layers, and tinged brown or bluish by iodine. There is no reason to believe that they agree with starch in composition.

The concentric bodies of the prostate are generally much larger, and probably have only a superficial resemblance to the corpuscles found in nervous tissue. Bodies of similar structure are met with in other parts, but their composition being entirely unknown, it is impossible to say whether they are identical with the corpora amylacea already described.

CHAPTER V.

NEW FORMATIONS.

It is difficult to give a perfectly exact definition of the class of new formations; for we shall exclude from it many productions which are not found in the healthy organism, and shall include in it some which are but the result of the action of parts normally existing. Thus we shall not mention the excessive production of fat cells, which takes place in general obesity, as an instance of new formations, while we shall consider as such the distension of a sebaceous follicle into an encysted tumour. This defect, however, is common to all arrangements. Nature presents us readily with distinct types of different classes; but rarely, if ever, does she define and separate her groups by any exact limitation, and the rigid taxonomist wearies himself in the search for that which does not exist. The idea which is conveyed in the term "tumours," seems, in a general way, most descriptive of the class now before us, which may be said to include all new *prominent* or otherwise *apparent local growths.* The character of growing excludes tuberculous and other deposits, and concretions. Thus an effused mass of blood even if it become organized, does not go on increasing in size. Tumours must also be distinguished from mere enlargements or hypertrophies in which no definite form is assumed by the newly formed elements. Finally they must be separated (though the separation is not always easy) from the products of inflammation. There is, doubtless, in the latter process a very abundant formation of new elements, but these do not compose any permanent structure. Either the result is something not strictly speaking organized at all (as pus), or else the organization is but transitory and preliminary to some higher kind of organization, as is the case with granulations. But since of some new growths it is also true that they tend to atrophy and decay after going through a period of progressive growth, it is clear that the distinction is one which cannot be absolutely insisted upon. The line is least clearly drawn in the case of two species of morbid growths sometimes classed as tumours, but sometimes as inflammatory products; and which we consider as a class apart—viz., tubercle and the products of syphilis.

Classification of New Growths.—New growths thus defined, differ very widely in their structure or anatomical characters; as well as in their effect on the organism, their mode of growth and increase. These differences supply two very different, but both important, bases of classification. (1) Anatomical structure. (2) Clinical or physiological characters, derived from the history and development of the growths. Proceeding on the first basis, we arrange new growths according as they resemble or differ from the normal tissues and elements of the body; on the second basis it has been customary to classify them as *malignant* and *non-malignant.* A third basis of classification is supplied by the nature of the tissue in which the new growth arises; the chief distinction being between those which arise from the class of connective tissues (including serous membranes) and those which have their origin in glandular or epithelial (including epidermic) structures. This distinction, which is founded on embryological development, will be further considered hereafter, but does not seem as yet to be sufficiently worked out.

In practical medicine the physiological properties are the most important. Without stopping now fully to define what is meant by malignant, we may say that this name is applied to tumours which affect many parts of the body at once, and which threaten the life of the individual, being associated with a general and fatal disease, not cured by extirpation of the part affected. On comparing the anatomical and physiological classifications, it is found that they do not by any means coincide. The property of malignancy belongs to new growths which differ very widely in anatomical structure, and it is consequently not possible, though it has long been the desire of pathologists, to define any one anatomical type which shall be absolutely characteristic of malignant tumours. Moreover the distinction of malignant and non-malignant growths, even apart from anatomical considerations, is by no means clear, no broad line can be drawn between the two; and the property will be found to be simply one possessed by different classes of tumours in unequal degrees. It will thus be possible to arrange the anatomical species of tumours in a graduated series, at one end of which will be placed those which are malignant in the highest degree, at the other end those which are not at all so.* From these considerations it is plain that the physiological characters cannot form a convenient basis of classification; and our arrangement will therefore be entirely anatomical; founded on a comparison of the structure of the new growths with that of normal parts.

Homologous and Heterologous Growths.—No new growth contains any elements essentially unlike those met with in the body either in its mature state or at some stage of development, but these elements are sometimes arranged in the same way as those of the normal tissues; sometimes in quite a different way; or else like the elements of normal tissues at an early stage of development. Proceeding from this basis, a classification of new growths as *Homologous* and *Heterologous*, has been proposed. Homologous growths are those which are like normal tissues of the body, heterologous those which are unlike normal mature tissues; those which resemble immature or embryonic tissues forming an intermediate class. But it should be understood that a new growth composed of simple and normal tissue may be heterologous if it occur in an organ of different structure and at a distance from tissue of the same kind (for instance, a cartilaginous tumour in the lung), and a homologous growth is, strictly speaking, one which arises by continuous development from a normal tissue of the body and resembles it in structure. Such a process of development is in fact, one of simple increase, and might be called hypertrophy; but if we regard the increase in number of histological elements as the most important fact, the term *Hyperplasia* will seem preferable; and thus all growths which are both in their origin and their ultimate form homologous may be called *Hyperplastic*. We can then use the corresponding term *Heteroplastic* for those growths, which are in the strict sense heterologous; that is, which are unlike the tissues from which they take their rise. It must be evident from what has been said that the production of hyperplastic tumours cannot be separated from the new growth of any simple tissue of the body out of tissue of the same kind. The product is called a tumour merely when it has a certain distinctness and isolation. We shall therefore consider the new growth of simple tissues under the same head as the corresponding tumours.

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* The chemical student will recall the division of the elements into electro-positive and electro-negative; a distinction which permits of their arrangement in an ascending or descending series, but not in two absolutely separate groups.

SIMPLE HISTIOID TUMOURS.

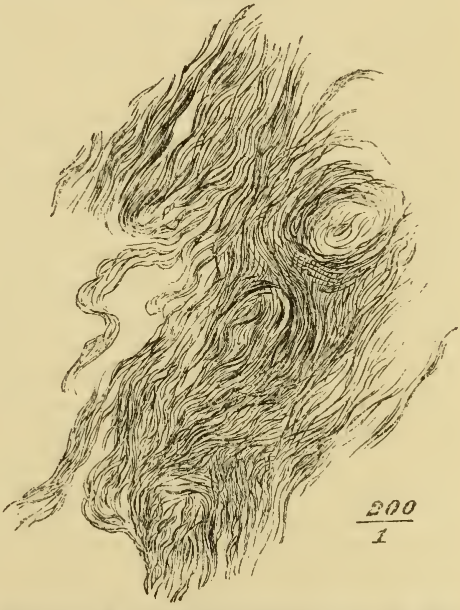
Those new growths which resemble the simple tissues of the body may be called *Simple Histioid Tumours*. This group includes the following species:—*Fibrous*, *Mucous*, *Fatty*, *Cartilaginous*, *Osseous*, *Vascular*, *Glandular*, *Muscular*, *Nervous*, and *Lymphatic* tumours.

I.—NEW GROWTH OF CONNECTIVE TISSUE; FIBROUS TUMOUR AND FIBRO-CELLULAR TUMOUR; FIBROMA.

The new formation of connective tissue takes place in a great number of pathological conditions. It is the most important factor in the process by which wounds are healed in vascular parts, and perhaps also in non-vascular parts; the same tissue often replaces special structures, such as nerve or muscle, when these are destroyed by violence or disease; and the production of connective tissue often constitutes a disease by itself as in the changes known as fibroid degeneration or induration, which depend upon an increase of the connective tissue normally existing in the organ affected. Newly formed connective tissue is usually found in connection with previously existing tissue of the same kind, and appears to be formed by continuous development from it; but in some cases it has been thought that it is produced by metamorphosis of the elements of the blood, either within the vessels as in the transformation of a blood clot into fibrous tissue, or outside in the case of white blood cells which have passed through the vascular walls. Connective tissue of new formation is usually well supplied and sometimes richly supplied with vessels. It is composed of fibres with connective tissue corpuscles and nuclei; the latter being especially numerous in recently produced tissue. It is liable to shrivel and contract; as well as to undergo both fatty and calcareous degeneration. When a new growth of connective tissue forms a distinct isolated mass it is either a fibrous tumour (*Fibroma, Innoma*) or a fibro-cellular tumour.

Fibrous Tumours.—These constitute a group with tolerably well-marked structural characters, but shading, almost imperceptibly, into other species of very different nature. They are essentially made up of fibres, more or less closely resembling those of connective tissue, but appearing in very various stages of development in different specimens. Sometimes the fibres are tolerably distinct and separate; more often so interlaced and blended together, or so imperfectly evolved, that they cannot be made out as such. Sometimes the fundamental substance is on the other hand amorphous or very indistinctly fibrillated. They contain also in various abundance the characteristic "connective tissue corpuscles" or plasmatic cells; which are stellate or fusiform, nucleated, and anastomose with one another. Yellow elastic fibrous tissue is not unfrequently mingled with the white. Beside these elements, fibrous tumours contain blood-vessels which are sometimes very numerous; but no other tissue in large quantity. Tumours com-

FIG. 16.



Section of a simple fibrous tumour of the skin. (From an original preparation.)

posed principally of smooth muscular fibre with some connective tissue, formerly called fibroid, are better classed with muscular tumours. The texture of true fibrous tumours is firm and tough, often grating under the knife; their surface is glistening and smooth; the colour white, reddish white, or greyish red. Little fluid is seen on the cut surface; what there is, is usually albuminous. They generally yield gelatine on boiling. In size they vary very greatly; some are extremely small, while some have been seen a foot or more in diameter, being among the largest of all tumours. They differ much in their degree of connection with neighbouring parts, being sometimes blended with these by continuity of tissue, but more often separated by a clear line of demarcation.

Fibrous tumours develop themselves in very different parts of the body; usually in such as normally contain much fibrous tissue. They originate from the skin and subcutaneous connective tissue of the trunk and extremities; sometimes in enormous number. Cutaneous fibrous tumours are sometimes pedunculated, and under this head must be placed the growths known as molluscum fibrosum. In other organs they almost always occur singly (if we leave out of consideration the so-called fibrous tumours of the uterus). They arise from fascia, from the periosteum (as in some forms of epulis); from the sheath of nerves (forming one kind of so-called neuroma); from bone; also in some glandular organs as in the kidney and mammæ; and genuine tumours of this class sometimes occur in the uterus.

Fibrous tumours are subject to several kinds of metamorphosis. Cretification is not unfrequent, and may either commence indifferently at any part, proceeding until the whole is converted into a calcareous mass, or it may be in great measure limited to the peripheral stratum, which it thus converts into a kind of shell, enclosing the rest. The calcareous degeneration affects tumours of very different size, and does not bear any relation to the age of the growth. The formation of true bone is less common, but has been observed in cases of fibrous epulis.

Pigment is sometimes abundantly deposited, and is said to proceed from antecedent hæmorrhage. Cyst-like cavities, filled with clear fluid, are occasionally found in fibrous tumours, constituting thus a fibro-cystic variety. Inflammation may occur, characterized by injection of the part, production of inflammatory cells, and sometimes by the formation of an abscess. Fibrous tissue is not unfrequently combined with some other growth in the formation of a tumour. In this way there result mixed forms, such as the fibro-fatty tumour, or fibroma lipomatodes, fibro-myxoma, fibro-myoma, and fibro-sarcoma. The signification will appear when we have described the corresponding forms of simple tumours.

Fibro-cellular Tumour.—This name is given to tumours composed of loose delicate forms of connective tissue, where the fibrous bundles leave considerable intervening spaces, filled with fluid.

This group is not very well defined, and there exists no sufficient reason for separating it from the true fibrous tumours.*

Clinical Characters.—Fibrous and fibro-cellular tumours may be taken as the type of "innocent" growths. They cause no ulceration or destruction of the neighbouring parts, except by mere mechanical pressure; they do not spread from one organ to another by contiguity; when removed they rarely return on the same spot; and cases of the production of similar tumours in a distant part of the body are of the greatest possible rarity. Two cases are, however, recorded by Sir J. Paget,† and others are referred to by Virchow.‡

II.—NEW GROWTH OF MUCOUS TISSUE, MUCOUS OR GELATINOUS TUMOUR, MYXOMA.

There is a particular form of tissue met with in some parts of the body, to which Virchow has given the name of mucous tissue. The only normal adult structure composed of it is the vitreous body of the eye, but it constitutes the Whartonian jelly of the umbilical cord; and in the embryo the subcutaneous tissue which afterwards becomes adipose, is at first of this kind. It is essentially a kind of connective tissue, and contains a network of stellate, anastomosing plasmatic cells. It differs from fibrous connective tissue in having a gelatinous semi-solid intercellular substance, containing in large quantity the ill-defined body mucin, which is dissolved by alkalies, and precipitated, without re-solution in excess, by acetic acid. This tissue occurs in pathological conditions chiefly as a distinct new growth or tumour, and hardly ever as a diffuse infiltration, unless it affect some other kind of tumour already formed. Tumours composed of mucous tissue or myxomata, are usually oval or round masses, tense and elastic when handled, and enclosed in a tolerably firm capsule of connective tissue. On section, they show a smooth, glistening surface, from which runs off, sometimes very copiously, a glassy or mucilaginous fluid. The consistence is that of "a flickering but tenacious substance, half pellucid, like size-gelatine." They bear some resemblance to œdematous connective tissue; only that the fluid which runs off is not serous, but contains mucin; and it never escapes so completely that the tissue collapses. The colour is usually yellowish, but may, from vascularity, or from hæmorrhage, pass through all shades of pink to a bright cherry red; and very beautiful mottlings, like those of a transparent pebble, are sometimes thus produced. Other portions are often white and opaque, either from the admixture of fatty tissue, or from the presence of very numerous round cells, which sometimes give the tumour a "medullary" appearance. On examining the fluid which escapes, or a small fragment of the tumour, blood globules will be seen, but in fresh specimens hardly, it may be, anything else, the cellular elements being singularly pellucid. On treatment with carmine, &c., or sometimes without, stellate or multipolar cells are seen, which are fragments of the cellular network. The tumour is seldom sufficiently solid to permit the cutting of a good section, but when this is possible the complete network of anastomosing cells is seen. There are always, beside these, round cells resembling those of embryonic tissue (or of granulations) which form no part of the network just spoken of. They exist in variable number, but when very numerous, produce a structure like that hereafter to be described as *medullary sarcoma*. Myxoma sometimes arises as a hyperplastic growth from the placenta, forming the well-known "uterine hydatids," which consist of an outgrowth of the mucous tissue, which the tufts of the chorion, equally with the umbilical cord, normally contain. In other cases the growth must be called heteroplastic, and arises from the neuroglia (or connective tissue of nervous structures), subcutaneous connective tissue, from intermuscular septa, from various fasciæ, from the sheaths of nerves, and from glandular

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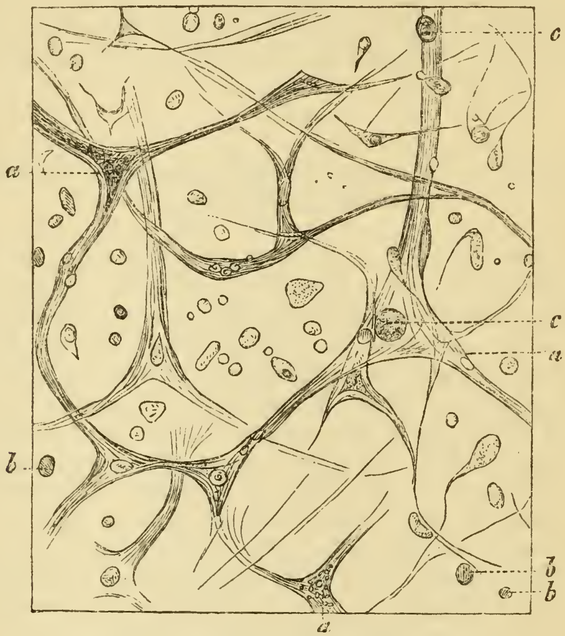
* The tumours called by Paget "fibro-cellular tumours properly so called," will here be described under the name *myxoma*.

† "Surgical Pathology," second edition, p. 762.

‡ "Die Krankhaften Geschwülste," i. p. 362.

FIG. 17.



Section of myxoma from the gluteal region.

a, Large stellate, or branched corpuscles, forming a network by union of their branches. *b*, Round cells, or free leucocytes. *c*, Similar cells, appearing as if enclosed in the larger corpuscles. Magnified 400 diameters. (Payne, "Trans. Path. Soc.," vol. xx., 1869.)

organs. Many examples of what is called neuroma come under this head, and many which were formerly called colloid tumours; also some polypi of mucous surfaces and the "fibro-cellular tumours, properly so called" of Sir J. Paget. Mixed tumours, in which this form is combined with some other kind of growth, are more common than the pure myxoma. Such are the *myxoma lipomatodes*, or combination with fatty tumour, where part of the tumour is composed of adipose tissue, into which the mucous tissue insensibly passes:* *myxoma cartilagineum* or myxo-chondroma, a tumour partly composed of cartilage. Cartilaginous tumours not uncommonly contain masses of gelatinous consistence, apparently derived from metamorphosis of their original structure, which are really identical with the structure of myxoma.† Myxoma fibrosum contains a large amount of fibrous connective tissue, and examples are also met with containing yellow elastic tissue. Myxoma is in general a perfectly innocent tumour, though it may recur after removal, or occur in a multiple form in the same tissue. There is no undoubted instance of its attacking different kinds of tissue simultaneously, or passing from one tissue to another by contiguity.

III.—NEW GROWTH OF ADIPOSE TISSUE; FATTY TUMOURS.

The production of adipose tissue, either as tumour and as a diffused mass, is a very common process. Local accumulations of fatty tissue are found in the interior or exterior of wasted organs (of which the kidney is the best instance), but can hardly be said to constitute a disease, except, perhaps, in the remarkable interstitial production of fat in muscles known as false muscular hypertrophy. General accumulation of fat, or general hyperplasia of adipose tissue, constituting corpulence or obesity, depends mainly on nutrition. Circumscribed productions of adipose tissue are the well-known fatty tumours.

Fatty tumours, or Lipomata, as they are sometimes termed, are of frequent occurrence. They consist of normal fat cells, closely packed together, and invested by a rather sparing quantity of common connective tissue. Occasionally this investment is more developed, and constitutes a kind of enveloping cyst; occasionally, also, it dips down, and forms a cystoid covering to separate portions of the tumour. They occur most often singly, but not unfrequently several exist together in the subcutaneous tissue. They attain, occasionally, an enormous size, so that records speak of specimens several feet in diameter, and weighing twenty to forty pounds.

Probably the largest tumour on record is a fatty tumour, of which a drawing is preserved in the Warren Anatomical Museum at Boston, U.S.A. It was much larger than the rest of the body of the unfortunate woman in whom it originated; and was estimated to weigh 275 lb. (actual weighing being impossible), while the body of the patient was estimated at less than 100 lb.*

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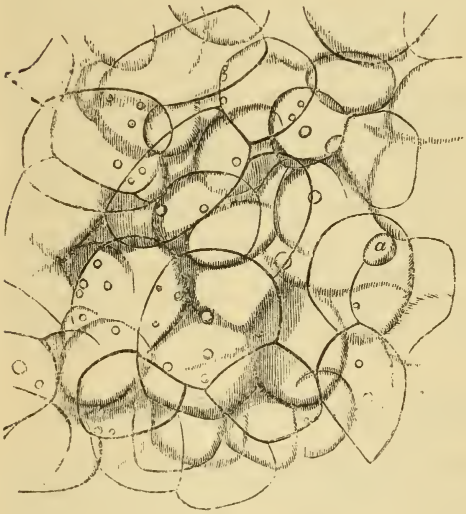
* See "Trans. Path. Soc.," vol. xx., p. 343. 1869.

† See Paget, "Surgical Pathology," second edition, p. 499.

Their most common seat is the subcutaneous connective tissue, especially in regions where fat is apt to collect, as on the buttocks, the thighs, the back, and neck, &c. They have, however, been seen in many other parts, as beneath the scalp, in the submucous tissue of the stomach, intestines, bronchi, and in the underlying connective tissue of the various serous and synovial membranes. In the knee-joint, especially, fatty growths have been distinguished by Müller as "lipomata arborescentia," in consequence of their branching form: this seems to result from their originating in the connective tissue, and growing inward towards the synovial cavity. Lipomata have further been observed in the lungs, liver, and kidneys, and in the bones. The surface of lipomata is commonly lobulated—their form, for the most part, globular; they have a peculiar doughy feel, with some degree of elasticity. They grow slowly, and occasion inconvenience only by the pressure they exert on surrounding parts: when at last this distension becomes excessive, the skin covering the tumour attenuates, and ulcerates, and a sloughing sore may be thus produced, which may destroy life by exhaustion. It sometimes happens that a fatty tumour seated in the submucous tissue pushes as it grows the yielding membrane before it, and thus, acquiring a pedicle, hangs into the intestinal cavity. A *steatoma* is a fatty tumour, with a preponderating excess of areolar tissue, and hence of firmer consistence.

Fig. 18.



Adipose tissue from a fatty tumour.

Fatty tumours are subject, though rarely, to calcareous degeneration, and may also undergo partial transformation into the tissue of myxoma. Formation of true bone in them is rare, but not unknown. They may also inflame and ulcerate.

Fatty tumours are strictly innocent; they may be multiple, but never produce metastatic growths in distant parts.

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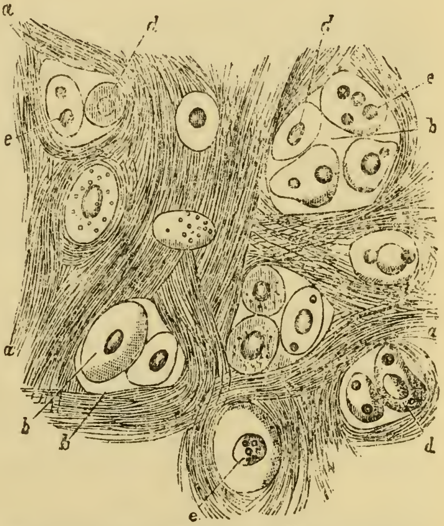
* Catalogue of the Warren Anatomical Museum. Boston, 1870, p. 642.

IV.—NEW GROWTH OF CARTILAGE; CARTILAGINOUS TUMOURS; CHONDROMA.

The pathological production of cartilage in a diffuse form may take place in the repair of fractures of bone;* in the formation of new or false articulations after dislocations, or as hypertrophy of normal cartilages, but never in the repair of cartilage which has become injured or destroyed. Outgrowths of cartilage, known as ecchondroses, are met with on the cartilages of the ribs, in the larynx or trachea, and on articular cartilages, such as the symphysis pubis. The dendritic vegetations of joints often produce true cartilage, which then exists in the form of pendulous masses. If these become detached by natural involution or accident, they form the loose cartilages, for which operative interference is sometimes necessary. Bodies of this kind are, however, not always cartilaginous, being sometimes fibrous and sometimes calcified. From these outgrowths must be clearly distinguished the true cartilaginous tumour (chondroma or enchondroma) which never grows from a pre-existing cartilage.

Enchondroma.—This name was applied by Müller to certain tumours essentially consisting of cartilaginous structure, whose real nature was first discovered by him. Enchondroma forms usually a globular tumour, with a smooth or somewhat tuberiform surface. Internally a section displays a structure which differs much in different cases, and is homogeneous, except in quite small tumours. Frequently some parts are gelatinous, or almost liquid, and others are composed of fibrous and vascular connective tissue. Some are composed of dense fibro-cartilage; in other cases this is replaced by an amorphous, firm intercellular substance, similar to hyaline cartilage. In fact, every variety of normal cartilage may be met with in these tumours; which occasionally present even the peculiar form of cartilage with stellate anastomosing cells found as a normal structure only in the cephalopoda.† The cartilage is in general arranged

FIG. 19.



Enchondroma composed of fibro-cartilage; microscopic structure. (After Lebert.)

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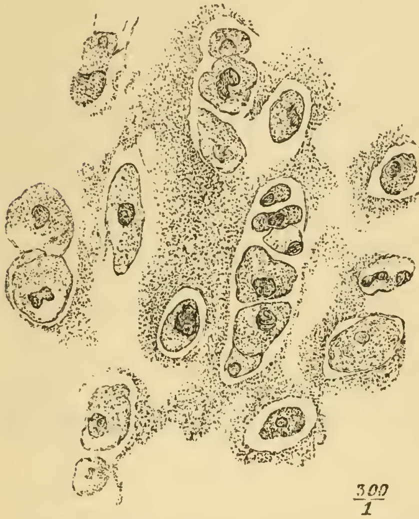
* Paget's "Surgical Pathology," second edition, p. 183; Wedl. "Pathological Histology."

† Paget's "Surgical Pathology," second edition, p. 495.

in numerous lobules, not larger than a pea or a bean, surrounded and separated by fibrous partitions, which contain blood-vessels. This arrangement illustrates the physiological law that a non-vascular tissue cannot be nourished at more than a certain distance from its blood-supply, and therefore cannot grow beyond a certain size. It is said that a cartilaginous lobule sometimes becomes vascular in its centre, and then grows, by the continuous addition of layers of cartilage to the outside; but this arrangement is certainly exceptional. The cells vary in size, from $\frac{1}{700}$th to $\frac{1}{1800}$th of an inch in diameter. They are sometimes simple oval or round cells, with clear nuclei; sometimes compound, sometimes with the double outline (showing the formation of a capsule) of normal cartilage cells; sometimes with several concentric layers; or again, they may have no obvious cell-wall, or even appear like free nuclei, which is especially the case in cartilage when about to soften. Stellate and ramified cells are rare, but very interesting, since they constitute a transition to the mucous tissue of myxoma. The intercellular substance is sometimes hyaline, sometimes more or less fibrillated, sometimes soft and mucous, and very various in amount. Enchondroma generally yields on boiling chondrin, like ordinary unossified cartilage, but sometimes gelatin.

Fig. 20.



Enchondroma from the lung, of pure hyaline cartilage. The growth was secondary to a similar tumour of the scapula. (From an original preparation.)

Three-fourths or more of all cartilaginous tumours arise from bone, sometimes in the interior (hence the name enchondroma), when they become surrounded with a kind of thin osseous capsule; more rarely on the outside, under or from the periosteum (perichondroma). The bones most frequently affected are the phalanges of the toes and fingers, with the metacarpal and metatarsal bones. In this situation the tumours are very often multiple. Cartilaginous tumours are also met with in the subcutaneous connective tissue, in muscular septa, fasciæ, and tendons; further, in glandular organs, as the testicle, salivary glands, ovary, and mamma. In the lungs both primary and secondary, or metastatic, tumours have been found. They most frequently affect young persons, and are occasionally congenital.

Enchondroma is of slow growth, and does not usually attain a large size; one instance, however, is recorded, in which the tumour weighed nine pounds and a half. It is subject to several kinds of metamorphosis; the cells may become fatty or calcified, and the basis-substance fibrous or granular, as in normal cartilage; or the whole may become calcified; or there may be, though this is not very frequent, a formation of true bone. Finally, the cartilage may undergo softening, a change due to mucous transformation of the basis-substance and fatty degeneration of the cells; the substance becomes gelatinous, honey-like, or pulpy, or may completely liquefy, and in this way cavities may be produced, and a truly cystic enchondroma result. If the tumour be near the surface there may be softening, leading to ulceration.

The combination of enchondroma with some other form of tumour is not uncommon. In or near the salivary glands, especially the parotid, tumours have often been described, which are in part truly cartilaginous, in part composed of the mucous tissue described above under the head of myxoma. They are distinguished from cases of simple softening of the cartilage by the form and arrangement of the cells. Such tumours may be called enchondroma myxomatodes. In this situation glandular or true adenoid structure may also be found. Combinations of enchondroma with lipoma, with sarcoma and with true cancer, have also been described.

Cartilaginous tumours were at one time thought to be without exception perfectly innocent; but, nevertheless, a few instances have been observed where a primary enchondroma was followed by metastatic growths in internal organs, especially the lungs. In some instances, as in the classical case of Sir J. Paget,* it is clear that the secondary tumours were produced by particles of the primary growth carried to the lungs by the circulation; but there is no proof that this is always the case.† A cartilaginous tumour has also been observed to extend itself by immediate infection from bone to soft parts, spreading along the connective tissue.‡ On the other hand, they have very rarely been observed to recur when once removed. From these facts the conclusion is that a cartilaginous tumour may be malignant, though the probabilities are very much against it. They do not produce any special cachexia, and produce inconvenience only by mechanical pressure, unless there should be ulceration, with profuse discharge.

V.—NEW GROWTH OF BONE; OSSEOUS TUMOURS; OSTEOMA.

The pathological production of bone may be a hypertrophy of pre-existing bone, in the regeneration of bone which has been destroyed; or a transformation of other tissues, especially cartilage and connective tissue; or an independent tumour. The immediate stimulus to the new growth may be repeated irritation or prolonged use, as in the ossification of tendons and fasciæ, wounds and consequent inflammation, independent inflammation, the "collateral hyperæmia" round diseased parts, the obscure causes summed up as senile change, or else some quite unknown cause different from these, as is usually the case with bony tumours. When bone is formed as a hypertrophy of tissue of the same kind, or to replace its loss, it is almost, if not quite invariably, formed from the periosteum; and it is in this membrane that the property, so to speak, of producing bone seems to reside, so that if the bone be removed, but the periosteum left, a perfect reproduction will take place; and even a portion of periosteum wholly or partially detached, will produce bone when transferred to another situation. This is remarkably shown in the modern *rhinoplastic* operation of transplanting, with a portion of skin from the forehead, a portion of the pericranium, so that not only a fleshy nose but a proper bony bridge is formed; as well as by the experiments of Ollier, who transplanted completely detached portions of periosteum to other parts of the body. The production of bone from connective tissue not in connection with a previously existing bone has often been confounded with simple calcification of that tissue, but in the following tissues undoubted bone has been observed,—in the tendons of muscles, in intermuscular septa, especially when the muscles are exposed to friction, as the inner muscles of the thigh, in riding, or the shoulder muscles in soldiers, when pressed upon by the musket. Serous membranes, as the pericardium and the pleura, are sometimes completely ossified. The dura mater and arachnoid of the brain or spinal cord sometimes contain bony plates. The hard plates in the interior of atheromatous arteries are probably never, certainly seldom, true bone; but, on the other hand, this has been seen in the gall-bladder and in the walls of hydatid cysts in the liver. The eye when atrophied from any cause, is very liable to produce bone, in the place either of the vitreous body or of the choroid. The production of bone from cartilage is seen most often in the costal and laryngeal cartilages; very rarely, if ever, in articular. The continuous outgrowth of bone, and the ossifications above spoken of are very difficult to separate from the bony tumours; in fact the difference is one of degree only. The principal outgrowth from bones which can be called distinct tumours are *exostoses* and *osteophytes*. Beside these we have to consider those which arise in an alien soil, or *heteroplastic osseous tumours*.

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* "Med. Chir. Trans.," xxxviii., 1855.

† For other cases, see Virchow, "Die Krankhaften Geschwülste," vol. i., p. 499 *et seq.* Also Virchow and Hirsch, "Jahresbericht der Gesammten Medicin. für 1869," vol. i., p. 184.

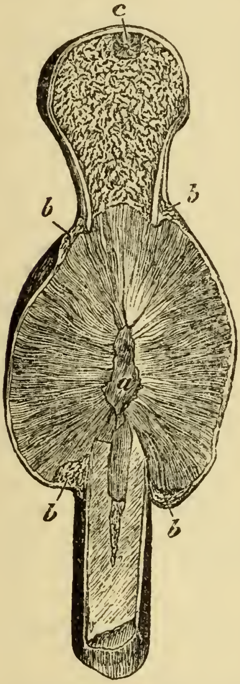
‡ Virchow. "Die Krank. Geschw.," vol. i., p. 490.

Exostoses.—An exostosis is an osseous tumour, proceeding from the bone or its periosteum, and, according to Rokitansky, homologous in texture, when fully developed, with that of its base and point of origin, whether that be compact or spongy. Its form varies—being sometimes broad and flat, sometimes round and prominent, with a narrow neck, sometimes spinous. Exostoses do not often attain a size above that of a hen's egg. Sometimes they are single; but often several exist together in the same individual, and even on the same bone. In texture they vary considerably from that extreme degree of density which constitutes the "ivory exostosis" to the porosity of ordinary cancellous tissue. The compact exostosis "is compact from the very first, and grows in such a way that the layers which are added to it always at once become as dense as ivory." "When they are minutely examined the number of peripheral laminæ is found to be very considerable, and the corpuscles lying amongst them are long. The Haversian canals are small, and far apart," and the corpuscles in some parts are quite absent, in others are closely crowded together. "The spongy exostosis proceeds from a circumscribed rarefaction or expansion of the bony tissue. It forms a tumour of cellular texture, abounding with marrow, which is surrounded by a compact layer or rind." It may originate from the compact outer layer of the bone, or from its spongy interior. In some cases a tumour of this kind contains in its interior a well-formed medullary cavity, communicating with that of the bone. The spongy exostosis may remain in the same condition, or become indurated and more similar to the compact, by the deposition of more earthy substance. If, as occasionally happens, a bony tumour grows inward into the medullary canal of a bone, it is termed an *Enostosis.* Exostoses often arise without any obvious cause; sometimes they appear to develop in consequence of a blow or strain. Sometimes they grow from many parts of the skeleton at once; as if from some constitutional cause. "In most cases the periosteum covering them is in its natural condition;" sometimes it is thickened and unusually adherent. They are not peculiar to any period of life; those of the spongy kind have been observed even in new-born infants. Spongy exostoses are sometimes destroyed by caries; and in a few cases the ivory exostosis has become necrosed, and been thrown off. This change must proceed, no doubt, from obliteration of the Haversian canals and consequent cessation of its nutrition, and would be similar to the shedding of the antlers of the stag.

Osteophytes.—To this class belong a great variety of bony growths, which form, for the most part, in inflammatory exudation, are pretty widely spread, in many cases, over a bone, and are rather easily separable from it. In these respects, and in their greater irregularity, they differ from exostoses, which are to be regarded as outgrowths from a bone, while osteophytes seem only to be produced under the influence of a bone. Osteophytes, of warty or stalactite shape, are very common in the neighbourhood of diseased joints, where the articular surfaces are affected with caries. They result, doubtless, from ossification of the exudation, derived from the adjacent hyperæmic vessels. In front of the vertebral joints and some other synchondroses, pretty long styloid or lamellar osteophytes are frequently produced, so as even sometimes to form a kind of bony capsule around them. "They arise from a chronic inflammation of the bones," and while these are atrophied, are often of dense hard texture. Sometimes the osteophyte is said to be foliaceous, consisting of beautiful delicate lamellæ, arranged parallel to each other, and running transversely vertical to the axis of the bone. The flat osteophyte is sometimes an extremely thin and delicate layer (like hoar frost); sometimes it is one or two lines in thickness. It is composed of delicate fibrils and lamellæ, and at first has scarce any attachment to the bone upon which it lies. "At a later period the osteophyte is found attached to the bone by some intervening minute round pillars and plates. After having become gradually compact it unites with the bone."

Heteroplastic Osseous Tumours.—Under this head come those instances of heteroplastic production of bone where the new growth is sufficiently distinct to be called a tumour. Their occurrence in tissues, naturally predisposed to ossification, as fasciæ and tendon, is not very surprising, and must be taken in connection with the continuous ossification above described. The same may be said of osseous tumours found in the neighbourhood of bones, and especially articulations, called by Virchow parosteal tumours.* But true circumscribed bony tumours have been seen in the lung, the brain, and the skin; for a fuller account of which we must refer to Virchow's work, already quoted.

Fig. 21.



Osteoid sarcoma of the femur, consisting chiefly of spindle-cells.
At *b b*, commencing ossification.
(After Virchow.)

Osteoid Tumours.—Beside the true bony tumours just described, there are others in which bone may occur. Some of these become osseous by the metamorphosis of fibrous or cartilaginous structure; but there is beside a distinct class of tumours growing from bone which themselves contain bone, and moreover give rise to tumours in other parts of the body having the same structure. They thus differ very much from simple osseous growths, and have accordingly been called malignant osseous tumours, or osseous cancers, or more recently osteoid sarcomata. A minute examination marks off some examples of this kind as being, in truth, cancers, of which the stroma has undergone calcification, and others as mixed forms in which cancer is combined with a bony growth; but there remain other cases to which neither of these explanations applies. These tumours always arise either from the inner layer of the periosteum or from the medulla of bone; but from their starting-point they may spread both into the bone itself and into the neighbouring soft parts, which, at first affected by a purely cellular growth, may at length become ossified. More generally, however, the tumour grows by addition of successive layers to the outside, and a very large mass, usually with a lobulated or irregular exterior, may thus be formed. The bony parts of the tumour are of a structure which, according to Sir J. Paget, is "true bone, but not well-formed bone;" according to others is merely a calcification of the intercellular substance; but it seems that both may be met with. The unossified parts of the tumour may be hard, but are principally composed of cells, in which the spindle form, characteristic of some forms of sarcoma predominates, but round cells and large cells of the myeloid type are also seen (Fig. 21). It is on this ground that Virchow and other recent writers classify these tumours under the head of sarcoma. Secondary tumours, precisely similar in structure to the primary growth, may occur in other parts, most frequently in the lymphatic glands; but sometimes, without passing through the lymphatic system, in the lungs. While in the former case there can be no doubt that the generalization is effected by the lymphatic channels, there is great reason to think that in the latter the secondary growths arise from portions mechanically transported through the veins and pulmonary artery.*

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* "Die Krankhaften Geschwülste," Bd. ii. p. 91.

VI.—NEW GROWTH OF BLOOD VESSELS; VASCULAR TUMOUR; ANGIOMA.

The Pathological Production of Blood Vessels.—Before we proceed to describe *vascular tumours*, which constitute our next class, we shall give some account of the development of new vessels in inflammatory exudations and other new formations. Two opinions have been held as to the mode in which their formation takes place. By some, as Paget, Travers, and Simon, they were considered to be formed by "outgrowth from adjacent vessels." Small dilatations appear on the side of a vessel, increase in length, and at last meet and coalesce with similar diverticula in their vicinity; in this way a new capillary loop is formed, and the process is carried on in the same way. On the other hand, Rokitansky, Vogel, and Dr. Walshe agreed in regarding the new vessels as originating spontaneously in the exuded blastema. Rokitansky has, however, given up this opinion so far as new growths and inflammatory false membranes are concerned, but still believes in the occasional endogenous production of blood-cells in certain cystic structures. With this exception, the belief in the independent formation of blood or blood vessels is now generally given up.†

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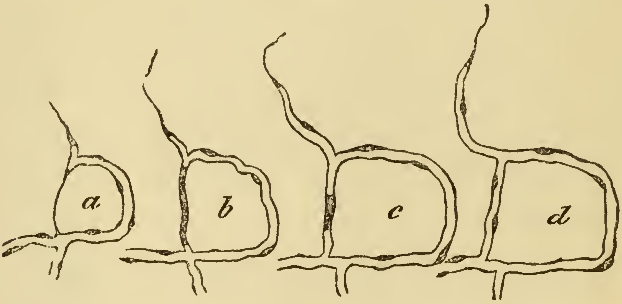
* Paget, "Surgical Pathology," second edition, p. 747.

† Billroth, however, in 1858, still attributed the origin of certain blood-corpuscles to the cells of the newly-formed vessels. Heitzmann, in a recent memoir ("Quarterly

According to Förster, there are two methods by which new blood vessels are produced. The first method is by the elongation of vessels previously existing. The capillary loops are drawn out to eight or ten times their original length, and when parts of contiguous loops come into contact with one another they unite. In this way a new anastomosing network of capillaries is formed, and the actual number of vessels is very greatly increased without any independent new formation. This process is seen only in hypertrophies of existing organs, in inflammatory growths, and in tumours. It probably corresponds to the normal method of increase of vessels in the growing organism. The second process described by Förster is the independent formation of blood vessels out of cells—that is to say, the cells of the connective tissue. These bodies sometimes proliferate, and the new elements thus produced arrange themselves in rows, till they at length form solid columns, or cylinders. These cylinders becoming hollowed out at the extremity by which they are in contact with previously existing vessels are converted into tubes, and permit the entrance of blood. In other cases a single connective-tissue corpuscle is the foundation of a new vessel. It enlarges till its prolongations become wide enough to admit blood corpuscles, and attaching itself to a previously existing vessel, establishes a communication with it. Thus the corpuscle itself, with its prolongations, becomes a capillary vessel, the walls of which are nothing but the original cell wall and its prolongation. This account can only be accepted by those who regard a connective-tissue cell as a space, and its prolongations as fine tubes or "plasmatic canals." It is also asserted that the connective-tissue corpuscles, with their plasmatic channels,

Fig. 22.



Formation of a new capillary vessel in the tail of the tadpole.

a, A vascular loop, partially connected by a protoplasmic thread. *b*, The protoplasmic thread become thicker and longer. *c*, The same partially hollowed out. *d*, The same converted into a hollow tube with two nuclei. The whole process occupied twelve hours. (After Arnold, Virchow's "Archiv," vol. liii.)

Journ. of Microsc. Science," 1873, vol. xiii., N.S., p. 169), believes in the endogenous production of blood corpuscles within inflamed bone and cartilage cells. To the same effect, Carmalt and Stricker, "Wiener Med. Jahrb.," 1871, p. 428.

may open up communication between two adjacent capillary loops, and thus increase the number of vessels. Still more recently the process of new formation of capillaries has been observed actually proceeding under the miscroscope in the tissues of living animals by Stricker, Golubew, Arnold, and others. Arnold observed the reproduction of a tadpole's tail, which had been amputated, and traced the series of changes represented in our figure. Solid protoplasmic buds were seen to grow from the walls of previously existing blood vessels, forming cords, and by their junction loops, which ultimately became hollowed out, and formed capillary vessels. The figure shows the changes observed in a single capillary outgrowth within twelve hours. Newly-formed capillaries are distinguished from those previously existing by their much greater width and often irregular outline. Their walls sometimes contain more numerous nuclei.

Vascular tumours are made up (it may be with other constituents also) of newly-formed blood vessels, or partly of previously existing blood vessels enlarged and altered. There are two kinds of them:—(1) Those composed of vessels similar to normal blood vessels, held together by more or less connective tissue; they are known by the names of simple vascular tumour, angioma simplex, telangiectasis, or angiectoma; (2) Those composed of a system of communicating spaces, with simple fibrous walls lined by an epithelium, similar to the cavernous tissue of erectile organs —known as cavernous tumours, angioma cavernosum, nævus cavernosus.

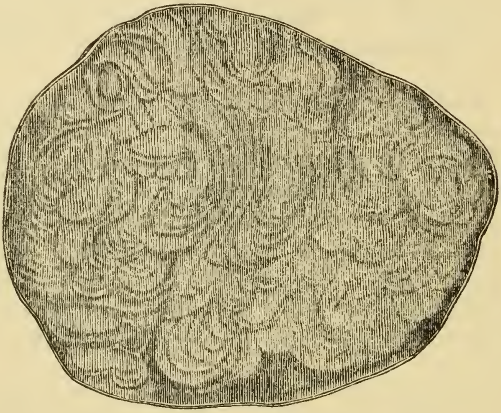
To the first class belong the ordinary nævi, either congenital or belonging to early life. These are flat, or slightly elevated structures, rarely distinctly projecting, which occur especially on the skin of the face, head, or neck, sometimes on mucous surfaces. They are composed mainly of contorted and convoluted capillaries, sometimes abruptly dilated or varicose, but not without small veins and arteries. Congenital nævi usually waste and pass away in the first months of life; but they may, by the multiplication of vessels and absorption of their walls, pass into the next or cavernous form.

Cavernous tumours are, however, more frequently of independent growth, though rarely congenital. They are alveolar in structure, being composed of a number of cavities communicating freely with one another in all directions. When cut into they usually collapse from the escape of blood; but if the blood should happen to be coagulated within them, they may form masses of very considerable weight and solidity. This cavernous structure appears to be always in communication both with veins and arteries, but not always by channels of the same capacity; the openings into the venous channels being generally much wider than the small branches by which blood enters from the arteries. The epithelium, by which the cavities are lined, has sometimes been mistaken for cancer cells. The tumours are sometimes clearly defined and incapsulated, but more often inseparable from the surrounding tissue. External cavernous tumours occur either in the skin or in the subcutaneous fatty tissue. Of internal organs they occur most frequently in the liver, where, however, they are by no means common. They have also been observed in the spleen, kidneys, suprarenal capsules, intestines, uterus, and ovaries, and on the inner surface of the bladder, and are sometimes multiple.* It is only in the rarest instances that they lead to hæmorrhage or any serious affection, and they are strictly innocent in their character. It is probable, however, that under the name of fungus hæmatodes instances of this disease have been confounded with cancer.

VII.—NEW GROWTH OF MUSCULAR TISSUE; MUSCULAR TUMOURS; MYOMA.

Of the two kinds of muscular fibre normally existing in the body, the striated is very rarely formed under pathological conditions. It is, however, produced for the restoration of muscles which have been wasted or degenerated by disease, and more rarely when loss of substance has resulted from a wound. In the latter case, muscle is often replaced by connective tissue. In the hypertrophy of muscles there is also a true new growth of muscular fibres. Tumours consisting of striated muscular fibre are among the greatest pathological rarities. The only cases in which this tissue composed the great mass of a tumour are those of a tumour from the testicle of a man aged eighteen, observed by

FIG. 23.



Section of muscular tumour of uterus.

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* For illustrative cases see "Trans. Path. Soc.," 1860, xi. 267; 1869, xx. 203.

Rokitansky, and two cases of similar tumours in the head of a fœtus. Striated muscle has in a very few cases been observed as a part of tumours of different structure.

New growth of smooth muscular fibre has never yet been observed in the restoration of lost parts or the healing of wounds. It undoubtedly takes place in the hypertrophies of structures which normally contain it, as in the enlargement of the uterus during pregnancy, and in hypertrophy of the prostate, the bladder, the coats of arteries, or the stomach.

Tumours consisting of smooth muscular fibre are most frequently met with in the uterus, forming the growths commonly called fibrous tumours.* They have recently received the name of *myoma*, or *fibromyoma*, or, for the sake of distinction from striated muscle, *leiomyoma*. These tumours consist of smooth muscular fibres, with variable proportions of connective tissue, which latter increases with the age of the tumours, the younger consisting mainly of muscular fibre cells. The characteristic anatomical peculiarity is, of course, to be found in these elongated cells; and though they have sometimes been mistaken for fibro-plastic or spindle-shaped cells, the distinction lies in the oblong or staff-shaped nuclei of the muscular tissue. These tumours are vascular, and they possess, from their muscular structure, a true contractility. They sometimes attain in the uterus an immense size, but after a certain age are very liable to calcification. They are met with also in other organs containing smooth muscular fibre, such as the prostate, the intestines, stomach, and in the walls of veins.

Muscular tumours are strictly innocent, though they often occur in considerable number; and almost the only inconvenience they occasion is that arising from weight and mechanical pressure. They are also very apt to cause hæmorrhage.

VIII.—NEW GROWTH OF NERVOUS TISSUE; NERVOUS TUMOURS: NEUROMA.

New formation of nerve tissue may take place as a restoration of destroyed tissue, or in the form of a distinct tumour. The healing of divided nerves is effected by the formation of new nerve fibres, which do not differ in structure from those previously existing. It appears also possible for ganglionic or grey nervous tissue to be completely restored, as in animals the lower part of the spinal cord has been regenerated when experimentally destroyed. A corresponding process has not yet been observed in man.

Nervous Tumour: Neuroma.—Tumours composed of nervous tissue are not very common; they most often occur in connection with the spinal nerves, more rarely on cerebral nerves, or on the sympathetic system. It is important not to confound them with fibrous or other tumours, which may be attached to or start from a nerve, though these have all been described under the common name of neuroma. The true neuroma is composed of nerve fibres, generally resembling those of the nerve trunk, to which it is attached, and therefore medullated or ordinary fibres. In rare cases, however, Virchow has observed tumours composed of the non-medullated fibres, or so-called fibres of Remak. The swellings which form at the divided extremities of nerves after amputation are instances of true neuroma, and similar tumours in the course of nerves sometimes have a traumatic origin. Isolated masses of ganglionic nervous tissue, in the form of tumours, have occasionally been found in the brain, especially in the walls of the ventricles: they are perhaps congenital. Ganglionic nervous tissue has also, in a very few cases, been found in the congenital cystic tumours of the testicle and ovary, and in composite sacral tumours; but these are doubtless instances of abnormal development rather than of what is commonly understood by disease. With these exceptions, nervous tumours never occur except in connection with nerve tissue; that is to say, they are never heteroplastic. They are also entirely without any approach to malignity, though they may be multiple.

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* Bristowe, "Trans. Path. Soc.," iv. p. 218.

IX.—NEW GROWTH OF GLANDULAR TISSUE, GLANDULAR TUMOURS, ADENOMA.

The tissue characteristic of secreting glands, or cavities lined with a secreting epithelium, may constitute the material of a new growth, and this may be either a hypertrophy of a glandular mass normally existing, or an independent structure. In the former case, which is by far the more common, the newly-formed glandular tissue resembles that of which it is an extension.

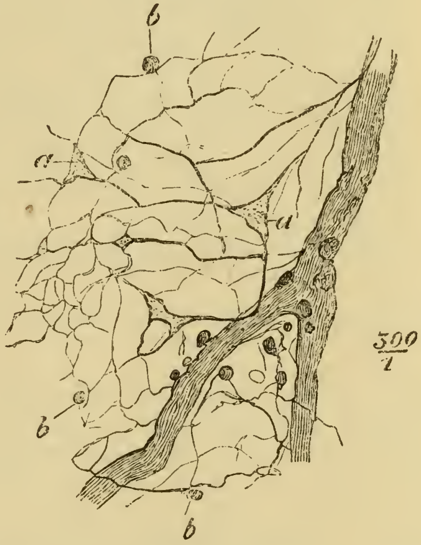
When occurring as a distinct tumour, the new glandular tissue still preserves the original type, and in some cases discharges the same functions as the original gland; though in other cases the excretory duct is deficient. Glandular tissue is very often a component of tumours which are formed mainly of some other tissue —as for instance the fibrous polypi of mucous surfaces, which often contain glandular tissue.

Perhaps the best example of this kind of tumour is the chronic mammary tumour, of which mention will be made hereafter, but similar structures are found in connection with the salivary glands, the liver, the follicles, and mucous glands of mucous surfaces. Glandular tumours never occur either primarily or secondarily except in close connection with secreting glands. The growth does not extend to the neighbouring parts, and they never give rise to other growths in distant and unlike organs, though they sometimes form an approach anatomically to the structure of true cancer. Their other peculiarities will be best described in connection with the special organs from which they arise.

X.—NEW GROWTH OF TISSUE RESEMBLING THAT OF LYMPHATIC GLANDS, OR CYTOGENOUS TISSUE; LYMPHADENOMA, OR LYMPHOMA.

The structure of lymphatic glands has of late years been very carefully investigated, and, as need hardly be mentioned, is found to bear no resemblance but in name to that of true or secreting glands. Its most important feature, or rather that which most concerns us here, is the presence in the follicular portion of a delicate fibrous network, or stroma, composed of fine threads, with occasional nuclei and corpuscles at their intersections, in the meshes of which the lymph corpuscles are enclosed (Fig. 24). While the same type of structure is preserved, there may be considerable variation in the relative proportion of the corpuscles and the containing network. The bands of the latter may even come to exceed in thickness the diameter of the corpuscles. The structure will then resemble what is represented in Fig. 25. This form of tissue is seen in lymphatic glands, which are the seat of chronic inflammation or the peculiar enlargement mentioned on the next page, and also in some new growths of cytogenous tissue. This peculiar type of structure, which has received the names of adenoid tissue and cytogenous connective-tissue, is not confined to lymphatic glands, but is met with in other organs not always regarded as belonging to the lymphatic system. The solitary and agminated follicles of the intestines, the thymus, and the tonsils are examples. This tissue, like others, is susceptible of hypertrophy or hyperplastic growth, and also of heteroplastic production in parts which do not normally contain it, and may in either case give rise to masses sufficiently distinct and permanent to be called tumours.

Fig. 24.

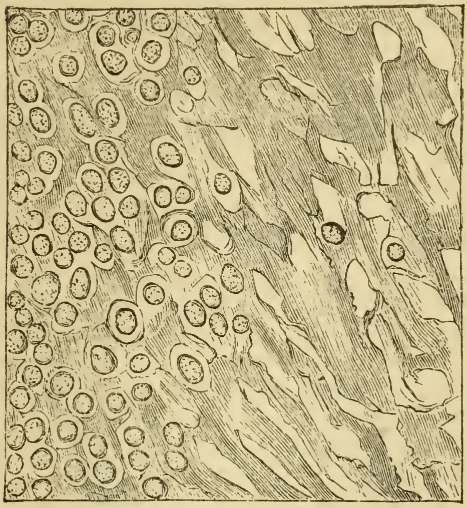


Stroma or network of a lymphatic tumour of the mediastinum.

a, Angular connective-tissue corpuscles forming part of the stroma. *b*, Free leucocytes or lymph corpuscles. A branched blood vessel is seen on the right of the figure. (From an original preparation.)

Hyperplastic Production of Cytogenous Tissue.—Hypertrophy of cytogenous tissue may occur in any of the situations where it is normally found. In the lymphatic glands this hypertrophy may attain considerable importance. Simple enlargement of these organs takes place either as a consequence of inflammation in the parts connected with them or independently; but the enlargement consecutive to inflammation, or other morbid processes, being usually single and not permanent, does not properly come under consideration here. Idiopathic enlargements of lymphatic glands usually occur in young persons, and very frequently end in a peculiar kind of degeneration called *scrofulous;* but as this process has a natural termination, and may completely pass away by suppuration or otherwise, scrofulous products cannot be regarded as strictly speaking new growths. The case is different, however, with another form of enlargement, in which no tendency to softening or caseous degeneration is observed, but in which the glands form hard, tough elastic masses, of a white or yellowish colour, and are usually adherent together in clusters. Simultaneously with this affection of the lymphatics are usually found new growths of cytogenous tissue in other organs, especially in the spleen, so that the process constitutes a general disease, to which we refer again below. Hypertrophy of the cytogenous tissue of the intestines is seen as a consequence of fevers and inflammatory processes, giving rise to enlargement of the solitary and agminated glands. It is also met with as an independent affection, in that disease in which the lymphatic system, spleen, &c., are usually also involved.

Fig. 25.



Cytogenous tissue, with thickened stroma. (Dickinson, "Trans. Path. Soc.," vol. xxi., 1870.)

Heteroplastic Production of Cytogenous Tissue.—Absolute new growth of cytogenous tissue is an accompaniment of a general disease usually starting with the lymphatic glands, which has received the names of anæmia lymphatica, adénie, and Hodgkin's disease. The lymphatic glands are always most conspicuously affected, and in the manner above described, but the new growths are almost as frequent in the spleen (where, however, it is difficult to distinguish them from hypertrophy of normal tissue of the same kind), and are also found in the liver, kidneys, intestines, lungs, &c.

The new growths are seldom distinct, but so intimately connected with the substance of the organ as rather to resemble an infiltration. They have much resemblance to the deposits of white blood cells met with in cases of leucocythæmia, but in the disease in question an increase in the number of white cells in the blood is not observed. Sometimes the newly-formed growth resembles the morbid form of cytogenous tissue previously described (Fig. 25), but sometimes the cells very greatly predominate.

Lymphoma and Lymphosarcoma.—Distinct tumours, composed of cytogenous tissue, unconnected with any general condition of that tissue throughout the body, are not very common. The most notable growths of this kind are seen in the mediastinum. Here they may arise from the lymphatic glands, or possibly from the remnant of the thymus gland. They sometimes remain isolated, sometimes infiltrate neighbouring organs. When a lymphatic tumour has the structure of a normal lymphatic gland, it is called a *lymphoma.* When the proportion of cells is very large, Virchow has applied the name *lymphosarcoma*, as making an approach to sarcomatous tumours. Many such tumours were formerly described as medullary cancer.

Clinical Character.—Most lymphomata and cytogenous growths are strictly innocent. Sometimes, on the other hand, they occur in many parts of the body at once, and are associated with a fatal cachexia: while even solitary tumours may grow very rapidly, and cause infection of the neighbouring parts. Tumours which possess these properties must be called malignant, though their structure is very simple, and quite different from what is truly named cancer. We must accordingly class these growths among those which are sometimes malignant.

We have now exhausted the list of new growths which can be strictly said to resemble any of the normal simple tissues of the body, and come next to those tumours which differ from normal parts either in their general form or else in their minute structure.

CYSTOID TUMOURS.

These constitute a very large and important class of new formations. They agree in the one general character, that they form receptacles which are filled with various contents, but in other respects they present very great diversity. We shall separate them at the outset into two divisions, the one comprising cysts which result from the distension of a natural pre-existing cavity, the other those which are entirely new formations. Under the first head we notice—(1) The common, so called *encysted tumours* which occur so often on the scalp, and elsewhere. These are essentially sebaceous follicles, whose orifice has become obliterated, and the cavity in consequence distended by continually accumulating secretion.

The contents of these cysts are of very different appearance, and the names of *meliceris*, *hygroma*, *atheroma*, *gummy tumour*, have been given to express a honey-like, watery, pultaceous, or jelly-like condition of the retained secretion. Examined microscopically, epithelial scales, free fatty matter, tablets of cholesterine, crystals of triple phosphate, and small hairs in various proportions, the epithelium, however, usually predominating, are found to constitute the contents of these cysts. The cyst itself appears as a thin, fibrous layer, lined on its inner side with epithelium. We have seen one case in which the epithelium in several tumours had accumulated in a very thick layer on the interior of the true cyst, giving rise to the appearance of a thick-walled cavity with contained matters of the ordinary kind; microscopic examination, however, showed that there was no real thickening of the cyst itself. The scaly particles of epithelium seem to fill themselves occasionally with a pellucid refracting matter, apparently of oily nature; they are mingled in some cases with granular globules, not unlike pus corpuscles, or if inflammation has occurred, with pus itself. This, at least, is the case with *comedones*, which are of similar nature. (2) *Mucous encysted tumours:* these are essentially similar to the preceding, and are formed by obstruction of the duct of a mucous follicle, or small conglomerate gland. They contain usually a glutinous mucous fluid. They occur in the lips, the mouth, in the cervix uteri, the Meibomian glands, and in the vagina.* *Ranula* is an exactly analogous affection of the duct of the sublingual gland. (3) Some of the renal and mammary and probably most of the hepatic cysts are produced in the same way, by local obliteration of the duct canals at two points, and distension of the intermediate portion. (4) Single cysts in the ovary are perhaps formed by dropsical distension of the Graafian vesicles; this may also be the origin of others which are afterwards compound. (5) Cysts in the thyroid gland are, no doubt, often formed by simple expansion of the normal vesicles. (6) Certain bursæ (not of new formation) become distended by a persistent increase of their secretion, and constitute cystic tumours. Mr. Simon states that the contents of these, instead of being fluid, are occasionally solid, the albuminous secretion having been replaced by a fibre-forming (probably fibrinous) blastema.

Under the second head we notice—(1) *Simple serous cysts*, and synovial bursæ. These arise in some cases evidently from the effect of pressure or friction, in others without any such cause. In the case of synovial bursæ we observe that a kind of condensation takes place in the areolar tissue of the part, marking out the limits of the commencing bursæ; within this the fibrous bands are gradually absorbed, while a secretion at the same time of fluid takes place, and at last the cavity is lined by a more or less perfect epithelium, and the new formation is complete. We can discern the purpose for which such cysts are formed, the end they serve, but we have no idea of the nature of the action which determines their formation. With regard to the others, which form in situations removed from pressure, as in the broad ligament of the ovary, the cause of their production is utterly unknown. They consist of a wall of fibrous tissue, varying in thickness in some measure according to the size of the cyst, condensed so as to form a smooth surface internally, and lined by a thin layer of epithelium, which has generally appeared to us to consist of nuclear particles, with imbedding granulous matter, and not of perfect cells. The import of this condition of epithelium appears to have reference to the rapid secretory action which takes place. The fluid contents of these cysts may be poor or rich in albumen, may contain abundance of cell forms, or very few, and may be either loaded with cholesterine, or devoid of it. We can confirm the statements of Mr. Simon, that the granule cells, which are sometimes very numerous, are the source of the colour of the dark coffee-ground-like matter which is sometimes present in large quantity.

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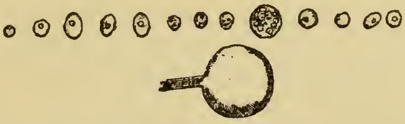
* We found the contents of an encysted tumour of the eyelid, operated on by Mr. White Cooper, to consist of a colourless, translucent matter, made up of multitudes of delicate granulous globules, imbedded in a clear fluid, which was coagulated in some measure by acetic acid.

FIG. 26.



Simple serous cyst, and epithelial particles from its interior—from vicinity of ovaries.

Some of these simple cysts contain quantities of fat, hair, teeth, and even bone, so that some good authorities have expressed their belief that they were the remains of a partially absorbed fœtus. This is certainly not the case, but their occurrence is of extreme interest, and when the growth is ovarian suggests very strongly that the normal reproductive function of this organ exerts itself by the development of these productions within its germ-bearing cavities, under the influence of some unnatural stimulus. This, of course, applies especially to the cysts alluded to under the first head, as developed from the Graafian vesicles, but it is Rokitansky's opinion that cysts of new formation may develop like products also. Sir J. Paget states, that in these cases the wall of the cyst acquires in some part the character of true skin, with hair follicles, sebaceous and sometimes perspiratory glands, and infers that "the structures and secretions formed on this portion of the cyst are shed into its cavity, and there accumulate, and that they remain, when, as often happens, the cutaneous structure on which they are produced has degenerated and disappeared." Several simple cysts may exist together in the ovary; this we should consider most likely to occur when they result from development of the Graafian follicles.

Cysts like those of the ovary, containing skin or its products (hair, fatty matter from sebaceous glands), and called by Sir J. Paget *cutaneous proliferous cysts*, are occasionally found in other parts, as the subcutaneous tissue, the testicle, lung, and other viscera, and even within the skull. They appear to be always congenital, and their origin has been explained by the supposition that at an early stage of development a portion of the integument becomes enclosed by the growth of the surrounding parts, and, finally, quite separated from the surface. In one very remarkable instance, a cyst containing teeth and bone was found in the anterior mediastinum. Simple cysts not unfrequently occur in the mammary gland, or rather in the dense areolar tissue investing it; they have a wall of condensed fibrous tissue, and according to Mr. Birkett, are lined by a characteristic epithelium, consisting of hexagonal particles. Their contents are either limpid, opalescent, non-albuminous fluid, or a tenacious, slimy, opaque, variously coloured, and concentrated solution of albumen. When combined with a peculiar growth of gland tissue advancing into their cavity, these tumours constitute the sero-cystic sarcoma, of which we shall speak more particularly when we come to the morbid anatomy of the mammary gland. Cystic formation may take place in various kinds of tumours, in fibrous, carcinomatous, sarcomatous, and may be a more or less prominent phenomenon.

(2) *Compound cysts.* The chief seat of these is in the ovaries, where they present two principal modes of development. In one of these the parent cyst, which for the most part continues to predominate in size, gives origin to a second generation of cysts, and then again to a third, and so on. The consecutive series of cysts are developed in the walls of their parents, but do not grow inward and occupy their cavities; the result is a multilocular mass, made up of numberless cysts which are filled with very various contents. These may be tolerably limpid and clear, or very viscous and greenish; may contain a very large number of celloid particles, or very few; may be variously coloured by blood-globules of new formation, or even replaced by a solid blastema loaded with blood corpuscles; fat, hair, teeth, and bone, may also occur in

Fig. 27.

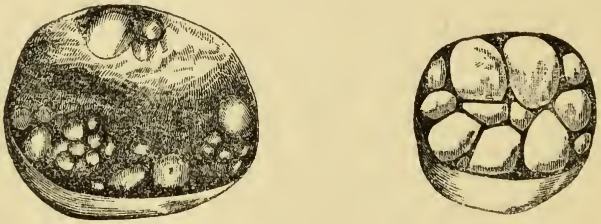


Diagram of compound cysts. In the left figure the secondary cysts are seen growing on the inner surface of the parent. In the right they have filled up the cavity.

these, as well as in the simple cysts. The partitions between the various cysts sometimes give way, and thus a tumour is produced, which internally seems imperfectly divided into compartments. In the other mode of development the secondary cysts grow inward into the cavity of the parent, which they fill up more or less completely; a tertiary race behaves toward them in the same way, and so on. This form may be combined with the preceding. Rokitansky describes a kind of villous, or cauliflower growth, which originates on the wall of the secondary or parent cyst, and may increase so as not only to fill the cyst cavity, but to break through its wall, and vegetate in the cavity of the peritonæum.* The impression left on the mind of the observer after a minute examination of the compound cysts, is, that they are of the lowest type of organization, resulting apparently from a depraved, degenerate formative action, which, withdrawing blastema from its proper uses in the system, hurries it with a wasteful expenditure into useless and injurious elementary shapes. How precious is the stringency of the law of our organic constitution which is comparatively seldom infringed by such terrible aberrations! It may be remarked, that the tendency to cyst formation most often appears in the existence of several together; it is certainly far more common to find several, whether of new formation, or resulting from distension of natural cavities, than to find a solitary one.

It should be mentioned here that the formation of cysts may occur in many kinds of new growth, both innocent and malignant, and in this way mixed forms result, such as cystic enchondroma, cysto-sarcoma, cysto-carcinoma, &c. The significance and general history of these growths will be determined by the structure with which the cystic growth is combined.

PAPILLARY GROWTHS, OR PAPILLOMATA.

These growths, which have also been called *epidermic* and *epithelial* tumours, constitute a well-marked class of new formations, which are of very frequent occurrence, and much practical interest. Warts and callosities of the skin are minor instances of this group, and consist simply in thickening of the epidermis, produced by accumulated layers of its scales. As an increased flow of blood to the part must take place it is not surprising that the papillæ of the corium beneath should, in some of the more advanced cases, become hypertrophied and elongated, so as to project upwards into the little tumour. In condylomata, mucous tubercles, and similar vegetations, which are apt to form about the orifices of mucous canals, under the irritation of syphilitic and other discharges, the surface is commonly observed to be lobulated or papillar, the interior marked by a vertical striation, while some vascular ramifications extend up into each papilla. The structure of these is beautifully figured by M. Lebert. The surface of each papilla, as shown in his plates, is formed by a layer of closely imbricated epithelial scales; while the deeper parts consist of either less flattened cells, or, according to our own observation, of nuclei, lying close together in a granulous and amorphous blastema. This interior nucleated granulous tissue, we believe, is continued downward to the base of the growth, and encroaches on the corium of the skin; for we have never been able to observe any clear demarcation between the vascular loops and the surrounding cell formation. Almost the very same description applies to those tumours which are most common on the lips, and whose cancerous nature one has often too much reason to suspect. These attain a much larger size, and are more manifestly vascular than the preceding, and their papillæ are more branched and grouped together, so that the surface resembles somewhat that of a cauliflower. We believe it is not always possible to say, from the structural characters of these growths, whether or not they are malignant, or whether, if removed, they will return again. In fact they pass by insensible gradations into the malignant form of epithelial growth, which we shall afterwards describe as *epithelioma*. Horns are epidermic productions, which are occasionally formed upon the head, the forehead, or some other part of the body. They originate in the sebaceous follicles, whose epithelium, thrown off in unnatural and excessive quantities, and mixed with the fatty secretion, forms a conical mass, which protrudes from the orifice in the skin, and is pushed onwards con-

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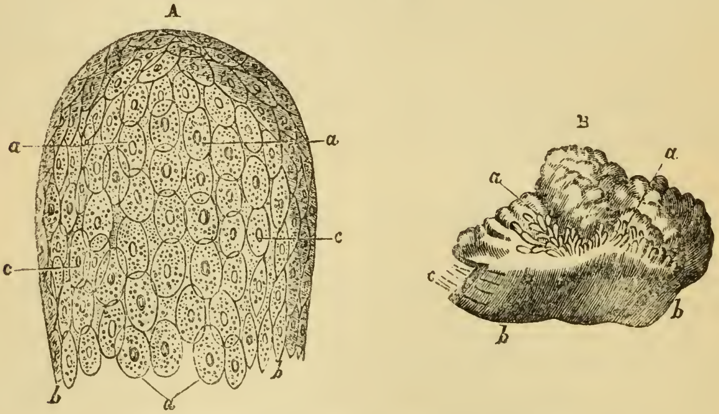
* In the Report of the Pathol. Society for 1851-52, there is an account, at p. 404, of a growth on the interior of an ovarian cyst which seems to be of this kind. It consisted of "vast numbers of pedunculated, clavate, clustered growths formed apparently of a simple basement membrane enclosing cells.'

Fig. 28.

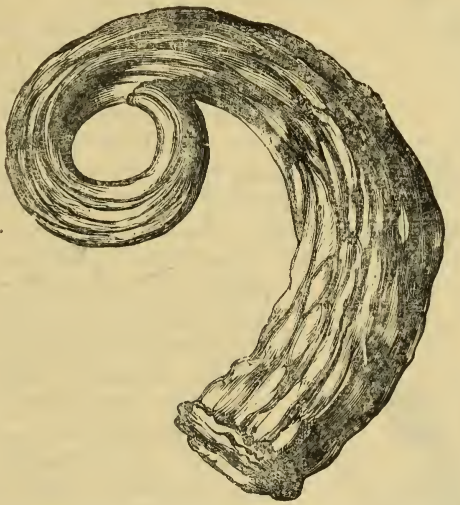


(A) Papillary prominence of epithelial growth.
(B) Epithelial tumour from lip.

(From Lebert.)

tinually by fresh accretions to its base. M. Lebert quotes a case in which the horn was six or seven inches broad at its base, and six inches long. A contusion, or ulceration of the skin, preceded the appearance of the tumour. A specimen was shown to the Pathological Society by Mr. Wagstaffe,* of a horn which had been growing for forty years on the leg of a woman aged eighty. It was of a spiral form, and if uncoiled would have been fourteen inches in length.

FIG. 29.



Horny growth from the face of a woman aged seventy-five, which had been growing twelve months. It started from an old wart, which had existed for many years. ("Trans. Path. Soc." xvi. p. 267.)

MELANOSIS AND MELANOTIC TUMOURS.

Melanotic tumours are not unfrequently spoken of by various authors, and occupy in their arrangements a place with other classes; but there is very great reason to doubt whether, properly speaking, any such thing ever exists. This is indeed generally admitted, the tumours thus described being really cases of melanotic cancer, melanotic sarcoma, &c. A few isolated cases are given by Virchow of simple melanotic growths occurring in situations naturally predisposed to pigmentation, such as the choroid, the pia mater, the skin, &c.; but he admits that all these are liable, when arrived at a certain stage of development, to assume a sarcomatous character. *Melanosis* (meaning thereby the deposit of black pigment) is an extremely common occurrence, and may take place in healthy tissues, in those which are variously diseased, and in new formations of any kind. The pigment is in the form of minute granules, or sometimes of almost dust-like molecules. It very commonly occurs free, though the particles may be more or less closely massed together, but is very often also contained in the interior of cells. There is nothing at all peculiar in these pigment-containing cells. They seem to be simply the natural cells of the organ, or of the new growth in which the deposit has occurred. Pigment granules are seen in the same part, both free and contained in cells, so that it is clear that their presence in the cells is of no special import. Rokitansky gives the following enumeration of localities in which the melanotic deposit takes place :—*In the lungs*, both in the air-cells (often in the epithelial particles), and in the connecting areolar tissue. When contained in the air-cavities it is in all probability chiefly inhaled as carbonaceous matter, floating in the air. We examined some time ago the lungs of a man who had been a worker in a gunpowder manufactory, and found them to contain a very remarkable quantity of free black matter. We are inclined to think the latter is not unfrequently the case, and for the following reasons :—(1) It seems quite impossible to believe that the very considerable quantity of black matter often found in the interlobular tissue of the lungs of old persons, should have proceeded from local congestions and extravasations, especially when there is no trace of previous inflammatory action. (2) We are well acquainted with the changes which hæmatine does undergo in the splenic tissue and in the renal tubules, when blood has escaped out of the vessels ; and we have far most commonly observed the colour of the granules to be an orange or reddish yellow. The same is the case in the remarkable production of yellow matter, which takes place in the congested centres of the hepatic lobules. (3) We have observed, particularly in the embryo of the fish, the development of pigment, and seen it commence by the appearance of a minute *free* particle of intense blackness, smaller than a nucleus, close by

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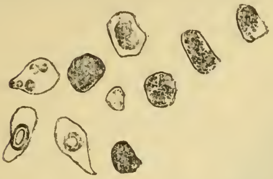
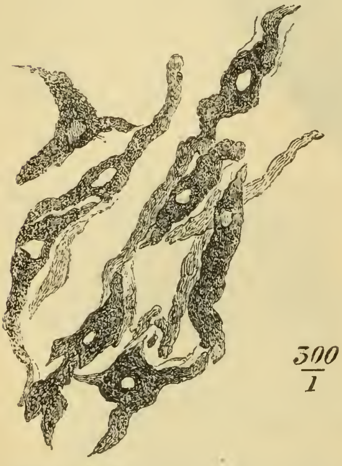
* "Trans. Path. Soc.," 1870, vol. xxi. p. 350.



Melanotic deposit in cells of an engorged lung. Some of the cells contain oil drops.

FIG. 31.



Branched connective-tissue corpuscles from the pia mater of upper part of spinal cord, containing much pigment. The specimen was from a case of Addison's disease, but only showed an exaggeration of the normal pigmentation of the part. (From an original preparation.)

the side of a vessel; so also in the abundant formation of black pigment which takes place in the liver of the frog at certain times there is not the least reason for regarding it as specially derived from the red colouring matter of the globules, but much more for supposing it to proceed from some of the highly carbonized matters contained in the liquor sanguinis.

Small particles of coal, having the peculiar "pitted" structure of coniferous wood, have been found in the interior of the lungs of coal workers by Traube. It has, however, been shown that the occurrence of minute hæmorrhages is another cause of the pigmentation of lungs.* In this instance, as in many others, truth lies intermediate between opposite opinions.

It also occurs in the *bronchial* glands, its quantity in them being usually in proportion to that in the lungs: it is here rarely contained in cells. In the *mucous membrane* of the stomach and intestines, of the *uterus*, and occasionally of the *air-passages*, it is generally the result of the irritation of chronic catarrh, and is derived from altered hæmatine. In the *mesenteric glands*, co-existing with such deposits in the mucous tissue, it produces a slaty grey, or still darker discoloration, spotted or uniformly diffused. In the *sympathetic ganglia*, in the *skin*, either naturally, in the dark races, or as the local discolorations, termed "melasma." In new formations, as intravascular coagula, atheromatous patches, and their cicatrices, in hæmorrhagic masses, false membranes of inflammatory origin, tubercle, especially the hæmorrhagic variety, colloid matter, and cancerous growths. Lastly, melanotic matter occurs in fluid exudations, and in the cavities of cysts. Rokitansky is of opinion that the derivation of the black pigment from the colouring matter of the blood is a settled point. We are rather inclined to agree with him than with Dr. Walshe, who thinks "that the relation of true melanic cell pigment to the constituents of the blood is altogether unknown." It is quite certain that, in all the instances above mentioned, except that of the air cavities of the lungs, the melanic matter proceeds in some way from the blood; but the question is, *how?* Is there first extravasation of the blood, exudation of its hæmatine, and conversion of this into the pigment, or is this produced from the liquor sanguinis, in some unknown way of secretion?

The chemical composition of melanotic matter is not accurately known, but is found to differ very much in different specimens. The black pigment of the eye was found by Lehmann to contain $\frac{1}{4}$ per cent. of iron. All analyses, however, show a large proportion of carbon—at least 50 per cent. It is quite insoluble in water, alcohol, ether, or dilute acids; but decomposed by strong nitric acid, and slowly dissolved by strong alkalies.

It thus appears impossible to separate a distinct class of melanotic tumours. Those to which this name is sometimes given will be described under the head of melanotic cancer, or melanotic sarcoma. Both may have malignant properties, but the former is a comparatively rare form of growth.

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* Rindfleisch. "Pathologische Gewebelehre," p. 356. First edition.

MALIGNANT TUMOURS.

In attempting to give a sketch of cancerous or malignant tumours, we think the best plan will be to take a typical specimen, which presents all the characters of the genus strongly developed, and to point out what these characters are. We shall afterwards notice the several species, and endeavour to show how the distinguishing features gradually become effaced, until the formation, as often happens, is, or appears, almost identical with those of a benignant nature. A tumour, of the species called encephaloid, is certainly the *παραδεiγμα* of cancer. It is of rapid growth, often attaining in a short time a very large size. Such a tumour is seldom solitary, but coexists with other similar ones in the same and in different organs. If extirpated it is sure to return, and probably diffuse itself more widely than before. It poisons the lymphatic current passing from it, and induces growths of like nature in the glands which that current traverses. It affects the general system with a peculiar cachexia, marked by languor, emaciation, debility, and a sallow complexion. It is very apt to infiltrate adjacent textures with its own substance, and, by absorbing their nourishment for itself, to occasion their atrophy; and, lastly, it tends, when exposed, to break down by a kind of decay, and to pour out profuse, exhausting discharges of serous, sanious, or bloody fluid. Such is cancer in its most malignant form. Of the above-mentioned characters, those which seem to us most nearly pathognomonic, are the tendency to infiltrate adjoining parts, to affect the glands traversed by the issuing lymph current, to reproduce similar growths in distant parts, and to return after removal. If these four characters are decidedly exhibited by any tumour there can be scarce any doubt of its malignant nature. It is to be remarked that all these characteristics are dynamic, and not structural. They result from the invisible qualities of the new formation, its mode of vegetation, dissemination, and reproduction, not from any peculiarity of form or arrangement of its particles. Whatever these might be, a tumour, which behaved as we have just described, would proclaim its malignant nature.

Experience has shown that these characters may be possessed by several kinds of growth, which differ very considerably in their general appearance and in their minute anatomy; so that no one type of structure is by any means peculiar to or characteristic of malignant tumours. There are, however, some structures which are almost certain to possess malignant properties, and others which are likely to do so; while in some forms of tumour (as the simple histioid tumours) malignancy is either quite unknown or exceedingly rare. The tumours which are *par excellence* malignant are those commonly called cancers, or carcinomatous tumours. We include under this head (1) encephaloid cancer; (2) scirrhus; (3) colloid cancer; and, (4) epithelioma; meaning by these names particular kinds of structure, anatomically definable. About these structures it may be said generally that they are almost sure to exhibit, either early or late in their course, the characters we have called malignant. There is a second group of tumours with respect to which we cannot make so absolute a proposition. They are very likely to be malignant; but, on the other hand, it is quite possible that they may remain confined to one spot, or be completely cured by removal. (Such tumours have been called *semi-malignant*, a term very objectionable, if taken to imply that they possess a peculiar property in a modified degree, but admissible, if meaning that they are less likely to be malignant than those tumours included in our first group). These may be called sarcomata, or sarcomatous tumours, and include the following forms:—(1) Spindle-celled sarcoma, or recurrent fibroid tumour; (2) round-celled, or medullary sarcoma; (3) myeloid tumour; with other less important forms. These two groups include all the forms of tumour in which malignant properties are common. The rare instances in which the history of the simple histioid tumours resembles that of malignant growths, have been sufficiently considered, when speaking of those tumours.

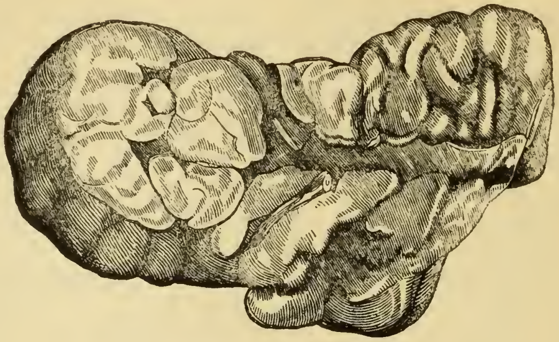
Since the sarcomatous tumours form a transition from innocent tumours to true cancers, it will be convenient to consider them first.

SARCOMATOUS TUMOURS.

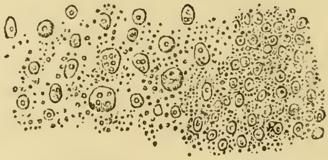
Sarcomatous tumours constitute a group, which it must be allowed, is somewhat ill defined. The fibrous group on the one hand, and the carcinomatous on the other, are the territories which border on the debatable land of sarcoma. The term may nevertheless very conveniently be retained as a collective expression for several classes of tumours, each of which, for itself, may be pretty clearly defined, and which agree in their history and mode of development as well as on certain particulars of minute structure. We shall describe under this head the growths known as fibro-plastic, recurrent fibroid, and myeloid tumours, with some forms of what were formerly called medullary sarcoma and others to be hereafter noticed.

Definition of Sarcoma.—Sarcoma may be defined as a growth chiefly composed of tissue resembling the immature or embryonic form of connective tissue. Such tissue is distinguished from adult or mature forms especially by the predominance of cells, so much so that it is a general, if not an absolute rule for fibre, bone, cartilage, &c., to pass through a stage in which their structure is entirely cellular. Though tissue of this kind constitutes the early condition of many new growths as well as of normal tissues, it is only in the growths under consideration that the development stops short at this particular stage. The definition of Virchow is substantially the same as this, but deserves to be quoted literally. "Sarcoma," he says, "is a formation whose structure belongs to the general group of the connective tissue series, and is distinguished from the clearly marked species of that group by the predominance of cellular elements." It will thus be evident that the structure of sarcoma cannot precisely resemble any tissue of the mature body, and cannot, therefore, be ranged with any class of what we have called histioid tumours. There is, however, another pathological product with which it has a considerable analogy—viz., granulations, which are produced for the repair of injuries and the restoration of lost parts; and since these are only the preliminary stage in the formation of a more perfect tissue, it is plain that their history and physiological position are also not dissimilar; the difference being that the condition which is temporary in the one is permanent in the other. Since the tissue of sarcoma is the common basis out of which the more special kinds of connective tissue may be evolved, it might seem likely that a further step in development should sometimes be effected, and one of these forms produced. This transformation does in fact not unfrequently

Fig. 32.



Sarcomatous tumour.



Structural elements of same.

occur, part of a tumour being strictly sarcomatous, while another part is composed of fibrous, bony, cartilaginous, or some other tissue. In this way mixed forms arise, which have been called fibro-sarcoma, osteo-sarcoma, chondro-sarcoma, &c.; the first part of the name being derived from the special kind of tissue with which the sarcomatous growth is associated.

The vessels of sarcoma are often larger than ordinary capillaries. and have thin or imperfectly formed walls, so as to appear like mere channels excavated in the substance of the tumour.

Origin of Sarcoma.—Sarcomatous tumours arise in the great majority of cases from some form of connective tissue, as the fasciæ and septa of muscles, the periosteum, the subcutaneous tissue (also from the skin), and in the brain and spinal cord from the peculiar connective tissue (*neuroglia*) which forms the basis of these organs. They do also arise in the interior of bones and in glandular organs as the mammæ, parotid, and also in lymphatic glands, but in the thoracic and abdominal viscera they are chiefly secondary formations.

The general appearance, form, and size of sarcomatous tumours differ so much in the different varieties that hardly any general assertion can be made about these particulars.

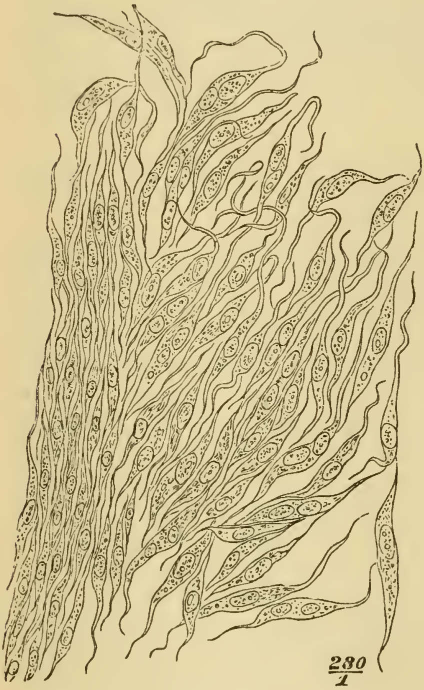
Structure of Sarcoma.—The elements composing a sarcomatous tumour are cells, intercellular substance, fibres, and vessels. The cells are either roundish, like lymph cells, spindle-shaped, and stellate (fibro-plastic cells), or sometimes irregular in shape and very large (myeloid cells). In general they resemble connective tissue cells and not epithelial cells. They contain usually one or two round or oval nuclei, with brilliant nucleoli; but the nuclei in the large myeloid cells may be very numerous indeed. The cells are often extremely fragile, so as easily to set free nuclei which may then appear to be the principal constituent of the growth. Hence the name of fibro-nucleated tumour given to some kinds of sarcoma. The intercellular substance is seldom or never absent, and sometimes abundant, though it may be inconspicuous. It is best brought into view by hardening the tumour, and is sometimes a very important character in the distinction of sarcoma from cancer. Fibrous tissue is found in the form of septa, dividing the tumours into lobes or capsules enclosing them, and the intercellular substance may also be fibrillated, but there is very rarely a true fibrous stroma forming loculi filled with cells, as is the case in true cancer.

The history of sarcomatous tumours is, in the majority of cases, that of innocent growths; but many examples occur of tumours which are in every respect as malignant as true cancer. This question is best considered with reference to the different forms of sarcoma.

VARIETIES OF SARCOMA.

Spindle-celled Sarcoma, Recurrent Fibroid Tumour, Fibro-plastic Tumour; Fibro-nucleated Tumour.—This is the commonest and best known form of sarcoma. It consists mainly of cells which, being elongated and tapering to each end, have been thought to show a transitional form between cells and fibres. Sometimes one or both ends are prolonged into two points instead of one. Similar cells are found in granulations which are on the way to form fibrous tissue, and in scars formed without granulations. The cells and nuclei have in general the characters described above; they vary very much as to size in different specimens, though within the same tumour there will be considerable uniformity; oval and round cells are almost always found associated with them. They are frequently arranged side by side in bundles, (as in Fig. 33) whence the name of *carcinoma fasciculatum* applied by Müller and Rokitansky to some tumours of this class. When such bundles, crossing one another at various angles, are cut in a fine section, the appearance of loculi or spaces may be produced, but there is not any true locular arrangement in sarcoma as here defined. These tumours arise most frequently from fibrous structures, as periosteum, fascia, muscular septa, &c., but are also found in glands, in the eye and other parts. They are often hardly distinguishable by the naked eye from fibrous tumours, but in other cases are translucent and soft, seldom opaque and "medullary" in appearance. Though mostly small they may attain the size of a man's head.

Fig. 33.

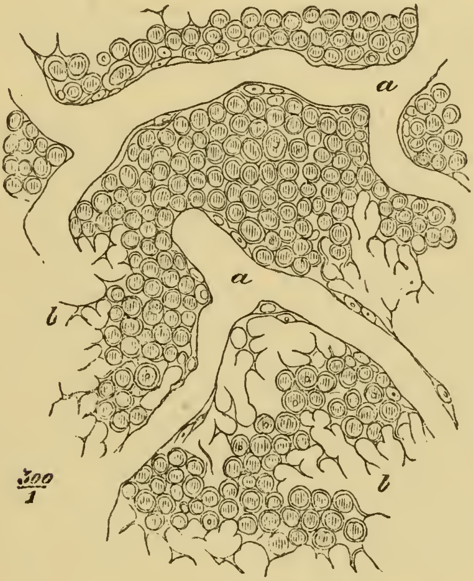


Section of spindle-celled sarcoma from the membranes of the spinal cord; partly teased out. The left hand of the figure shows the fasciculate arrangement of the spindle-cells. On the right hand they have been separated from one another, to show their tapering prolongations and large nuclei. (After Virchow, Die Krank. Geschw. vol. ii. p. 197.)

Clinical character.—Tumours of this group may often be completely and finally removed by operation, and produce no general affection of the constitution: but very often a new growth takes place at the seat of operation, and the tumour may in this way recur several times. It may also give rise to growths in distant parts of the body, especially the lungs ; and the history of such cases may be in no respect different from that of cancerous tumours. We must therefore consider it as among the possibilities with regard to a spindle-celled sarcomatous tumour that it may produce all the phenomena of malignancy ; though statistics would show that this event is by no means so likely to happen as in the case of true cancer.*

Round-celled Sarcoma; Medullary Sarcoma; Embryoplastic Tumour.—These tumours have been in many cases described as medullary or encephaloid cancer, which form they often resemble both in their external appearance and in their history. Their characteristic structural forms are roundish or oval cells, enclosing one or more nuclei, but presenting little diversity of form. The size is generally comparable with that of lymph cells, but may be larger. The cells are separated by more or less intercellular substance, though the amount of this may be trifling. In external appearance, they vary very much as to size and form. On section they appear when fresh for the most part slightly translucent, but soon become opaque, or as it is said " medullary ; " and this medullary appear-

FIG. 34.



Section of round-celled sarcoma. *a*, thin-walled vessels ; *b*, masses of roundish cells, with a little intercellar substance in some parts, made evident by hardening. (After Rindfleisch).

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* It has often been noticed that lymphatic glands are spared by sarcoma, and that metastasis or infection takes place independently of these, but this must not be taken as an absolute rule.

ance may prevail even from the first. If examined some time after death a distinct "juice" may be obtained by scraping, which is composed of cells set free by incipient decay. It would be rash to say that this juice is never seen in recently extirpated tumours. The colour depends principally upon the greater or less amount of vascular supply. These tumours show a greater tendency than spindle-celled sarcomata to softening and ulceration; cavities containing milky liquefied portions often occurring in them. Their most frequent situation is in the central organs of the nervous system, but they may occur in almost any part.

Clinical character.—They are very likely to recur after removal, to affect neighbouring organs by contiguity, and more frequently than any other form of sarcoma to produce secondary growths in the internal organs. They may thus often deserve the name of malignant, but, nevertheless, the prognosis will on the average be more favourable for a tumour of this description than for one having the anatomical characters of cancer, to be hereafter described.

Alveolar Sarcoma.—Tumours are sometimes met with having the clearly marked alveolar structure, which will be described below as characteristic of cancer; but in which the cells have no pronounced epithelial character. Considering that this form of growth is sometimes found continuous with true sarcoma; that it may arise from connective tissue, and that its cells have an indefinite character, some writers (as Billroth) regard them as sarcomata. Others again would call them "connective-tissue cancers;" and in the classification here adopted they must necessarily come under the head Cancer.

Myeloid Sarcoma; Myeloid Tumour; Sarcoma with Gigantic Cells.—In all forms of sarcoma, as above defined, there are occasionally found very large, many-nucleated cells, of irregular shape, but these cells are found in greater abundance, so as to form a characteristic structure in the class of tumours to be now described, which was first distinguished by Sir J. Paget. They are termed *myeloid*, from the similarity of their structural elements to certain corpuscles found in the marrow (*μυελόν*) of young bones. The tumour may either be enclosed in a capsule of expanded bone, or only by a periosteal investment. They are, for the most part, rather firm, but brittle, compact, of spherical or ovoid shape when invested by bone, more irregular and lobulated when growing upon bone. The cut surface is smooth, uniform, shining, succulent, with a yellowish fluid; commonly variegated by blotches of dark or livid crimson, or various shades of red upon a greenish or grayish white ground. The structural elements characteristic of myeloid tumours are large, round, oval, or irregular cells, and cell-like masses, of clear, or dimly granular substance, $\frac{1}{300}$—$\frac{1}{1000}$ inch diam.; and containing from two to ten or more oval, clear, and nucleolated nuclei. With these occur caudate and fibre cells, and free nuclei, and the whole are embedded in a dimly granular substance, mingled with more or less of molecular fatty matter. The history of these tumours, as far as is known, is nearly as follows:—They usually occur singly, are most frequent in youth, and very rare after middle age; they are of slow growth, cause no pain, and have no tendency to ulcerate.

These tumours occur usually in connection with bone, and many of the growths on the jaws called epulis are of this kind, but instances are not wanting of their occurring primarily in other parts, as the eye, the breast, and the peritoneum.* Such cases are, however, pathological rarities. Myeloid tumours seldom grow again if completely extirpated, but local recurrence is always possible, and in a few cases the production of secondary growths in the internal organs has been satisfactorily demonstrated. It is impossible to refuse to such cases the epithet of malignant, but in general a favourable prognosis may be formed as to a clearly marked myeloid tumour.

Melanotic Sarcoma.—Some specimens of sarcoma, both of the round-celled and the spindle-celled varieties, are remarkable for the amount of pigment contained in them. They are among the tumours which have been described as melanotic cancer. Such forms originate most frequently from those parts of the body where pigment naturally occurs, as the skin, the choroid coat of the eye, &c. Many of them, especially those from the eyeball, are in the highest degree malignant.

Cysto-sarcoma.—Cyst formation, combined with sarcoma, constitutes *cysto-sarcoma.* Enlargement of the natural cavities and channels, such as glandular acini or ducts, seems to be the process by which the cysts originate. Cysto-sarcomata mostly occur in the female breast and in the ovary, and the special forms belonging to these organs will be discussed in the special part of this work. It may be laid down as a general rule that such tumours are sarcomata in which a cystic growth occurs, and that this growth does not alter their general characters or probable history, which will be that of the particular species of sarcoma to which they may happen to belong.

Psammoma.—We may also here allude to a class of tumours peculiar to the brain and its appendages, which are usually classed under the head of sarcoma. Their characteristic feature is the occurrence of calcareous matter, or "brain sand," in the centre of small concentric lobules. They are not uncommon, and seem to produce, under ordinary circumstances, no symptoms whatever; it is only when they attain an excessive size that they have any pathological importance. Their consideration comes more properly under the head of the special pathology of the brain.

Glioma.—This seems the proper place for giving some account of a class of tumours peculiar to the brain and similar nervous structures, which are formed on the type of, and in fact developed from, the interstitial substance which forms the connective-tissue matrix of the nervous centres, and to which Virchow has given the name of neuroglia. A hyperplastic growth of this substance, sufficiently distinct to form a tumour, constitutes a glioma. This form of tumour is undoubtedly very nearly allied to sarcoma, and indeed it is doubtful whether any clear line can be drawn between them. Gliomata generally occur as solitary tumours, more rarely multiple; they vary much in size, and may be as large as a fist. They are sometimes extremely vascular, but sometimes again almost non-vascular, pale, and of a hardness approaching that of cartilage. They consist chiefly of round or somewhat polygonal cells, separated by a very distinct, but homogeneous matrix. They are very liable to fatty or caseous degeneration, so that they may sometimes approach the consistency of yellow tubercle. On the other hand, the degeneration sometimes results in the production of a cystic cavity. These tumours occur in the immense majority of cases in the brain, but similar structures have been seen on the nerves of special sense, and in connection with the retina. They rarely spread by infection to neighbouring structures, and there are very few instances on record of the occurrence of secondary tumours in distant parts. They may therefore be spoken of as generally, though not universally, innocent.

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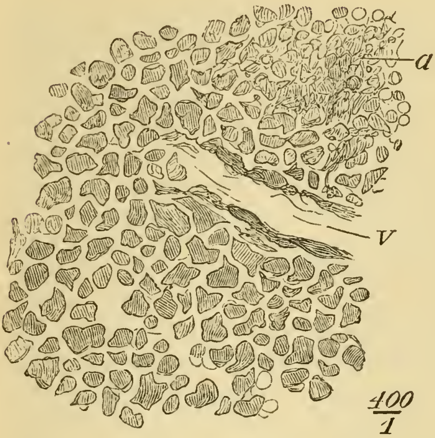
* Virchow. "Die Krankhaften Geschwülste," ii. 336.

Fig. 35.



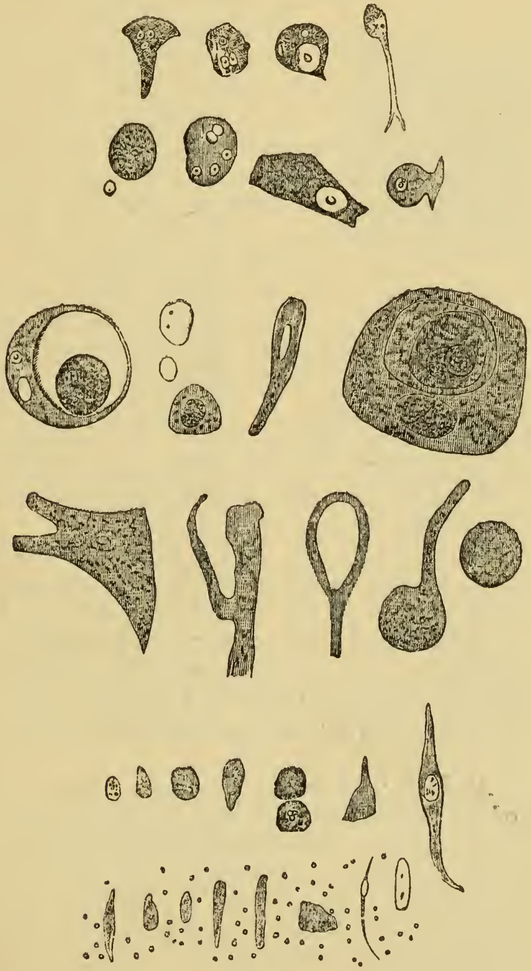
Section of a glioma from the brain, showing the cells or corpuscles imbedded in a clear matrix. *v*, a vessel. *a*, portion of the tumour where degeneration and caseation have begun.
(From an original preparation.)

CANCERS OR CARCINOMATOUS TUMOURS.

There are three varieties of cancer which agree to a certain extent in their structure, and about which, therefore, certain general propositions may be made. In each of these forms (encephaloid, scirrhus, colloid) there is a fibrous framework or stroma arranged in such a way as to form alveoli, or spaces communicating freely with one another, in which are contained cells, closely set together and not separated individually by any further partitions, or any intercellular substance. This is the only anatomical character common to all the tumours which we call cancer, and it serves, in the immense majority of cases, to distinguish them from the sarcomatous tumours which most nearly resemble them. The cells vary much; but in the majority of cases they are angular and flattened—that is, they have what is called an "epithelial" character.

Encephaloid or Medullary Cancer.—This is commonly called soft cancer in distinction from the form to be next described, scirrhus or hard cancer. Its softness depends upon the great preponderance of cells, and the comparatively slight development of the fibrous stroma. Medullary cancer may occur as a diffuse

FIG. 36.



Encephaloid—The first and the three last of the sets are from the liver, the second is from a bone, and the third from the vertebral column. The great difference of the cell forms is very apparent.

infiltration, or as a distinct tumour; even in the latter case it is usually very closely connected with adjoining parts, and surrounded by no capsule, or a very ill-defined one. The external form, the soft consistence, and sometimes the colour have been thought to resemble the brain, whence the name encephaloid. On section a thick creamy "juice" is generally seen, either exuding spontaneously or on pressure, or else obtained by scraping the surface with a knife. This juice contains, or is composed of, cells set free from the alveolar spaces; and if they be all removed from a thin slice of the cancer, the amount of fibrous tissue left is sometimes very small. The stroma is, however, never wanting, and has the alveolar arrangement already described. The substance is opaque, and in colour sometimes pure white, but more often modified into various shades of red by the presence of blood vessels. The cells of medullary cancer are very difficult to characterize. They vary much, both in size and shape, and when large they are mostly flat and angular, or polygonal, so as to have an "epithelial" character, which, if well marked, is very distinctive. Such cells contain one or several nuclei, sometimes themselves larger. In some soft and rapidly-growing cancers, the cells are very small, and have in this case a much less pronounced epithelial character, but still show great diversity of form. There are also often free nuclei to be seen, which are possibly derived from fragile and easily-destroyed cells. In tumours so subject to softening and fatty degeneration as medullary cancers, all cell forms are liable to be destroyed, and a comparatively young portion of the tumour should therefore be sought for.

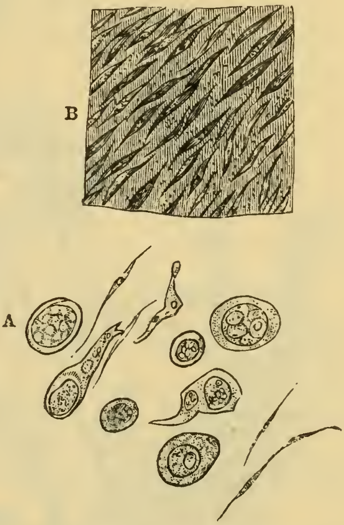
History of Medullary Cancer.—This form of cancer presents, as has been said, in a very marked degree the characters above described as malignant. It is, however, worthy of note that its growth is, as compared with other forms, extremely rapid, and that it is by no means specially a disease of advanced life. It is most frequent between the ages of forty and fifty, but may occur in infancy, and cases of cancer in youth are almost invariably of this variety.

Varieties of Medullary Cancer.—Various names have been given to varieties of medullary cancer which differ in their consistency and general appearance. More important characters are denoted by the following names:—The term *Hæmatoid* implies that the growth is unusually vascular, is the seat of excessive development of vessels, and perhaps of blood, and is prone to pour out those alarming hæmorrhages which often cause fatal exhaustion. When the hæmatoid character is strongly marked, the name of Fungus Hæmatodes is applicable. *Melanoid* cancer is in the great majority of cases encephaloid structure, with the addition of black pigment. The secondary growths which follow a primary melanoid cancer may be unpigmented.

Villous Cancer is a growth of medullary cancer projecting into a cavity, such as the bladder, where it takes a villous or polypoid form; but epithelial and other growths have been confounded under the same name.

Scirrhus, or hard cancer, commonly appears as a knotty or uneven, pretty distinctly limited, very hard (stony) tumour. Its surface or section is of a bluish or greyish white, and often presents a peculiar glossiness; scarce any trace of vessels is ordinarily visible, except in spots, which are inflamed and softening. The adjacent tissues, especially the skin, when the growth is subcutaneous, are more or less involved, and drawn inwards towards the tumour. In structure it consists essentially of a basis substance, more or less advanced in fibre-development, in which very various forms of cell growth are imbedded. Of the latter, it is utterly impossible to give any general account; they may be bare nuclei, cells of most various aspect, vesicles; granular globules; with these oil and diffused granulous matter are mingled in varying quantity. Glomeruli are often seen in fattily degenerating, or in inflamed parts; and parent cells, containing a secondary generation, are occasionally present. The disposition of the fibres is very various, sometimes parallel to each other, sometimes radiating, often crossing at right or acute angles. An alveolar arrangement may, however, be regarded as the prevailing one in almost all specimens of scirrhus at the height of its development, and the alveoli are filled, as in encephaloid, with cells of very various shape and size. The chief difference observed on comparing these two forms of cancer is the greater predominance of cells in encephaloid, and of the stroma in scirrhus.

Fig. 37.

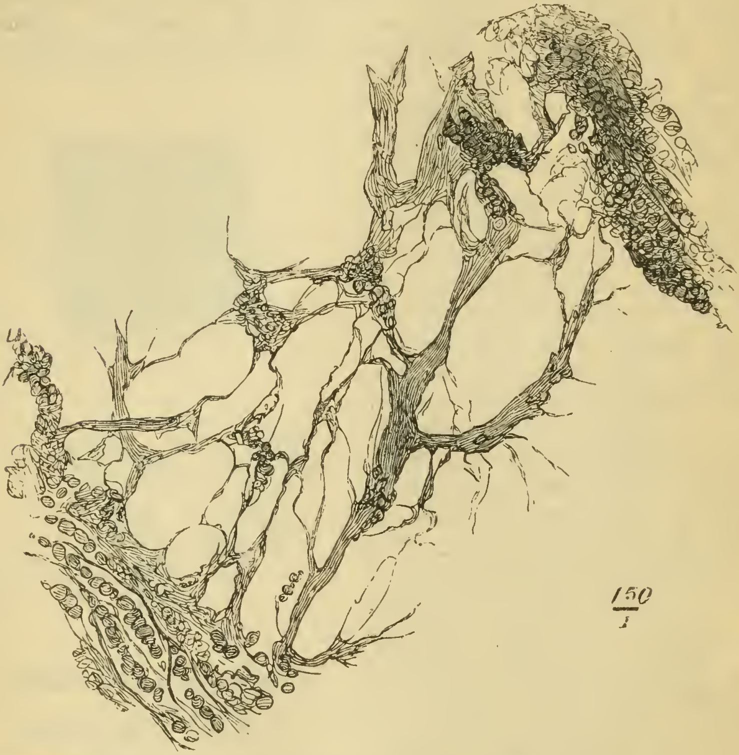


Scirrhous tumour of cerebrum.

(A) Cells.
(B) Section of firm stroma.

In atrophied, or wasted specimens of scirrhus, the alveolar arrangement is often lost, and the cells are quite unrecognizable. Scirrhus yields some gelatine on boiling; less albumen and oil, but more saline matter, are contained in it than in encephaloid. The growth of scirrhus is slow, the more so in proportion as its fibrous element predominates; it may then exist long without inducing the constitutional cachexia, or reproducing itself in any distant part, or even affecting the lymphatic glands. The most common seats of scirrhus are the female breast, the pyloric extremity of the stomach, the rectum; it is usually the original formation in these, or in other parts; but gives rise to secondary growths, either similar to the original or encephaloid. Often it extends itself by infiltration among adjacent tissues. It rarely attains a large size, and is not often seen so large as an orange.

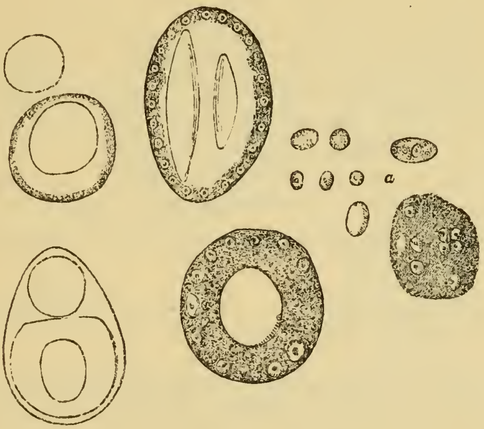
Fig. 38.



Fine section of scirrhus of the pleura, from which the cells have been washed out to show the alveolar stroma, composed of bands varying in thickness. To the left of the figure may be seen the development of alveoli. (From an original preparation.)

Colloid cancer, the next variety we notice, is also called alveolar; other cancers present more or less of an alveolar arrangement, but it never constitutes so prominent a feature of their structure. The walls of the alveoli consist of a fibroid tissue, sometimes extremely delicate and translucent, sometimes, and especially in the deeper layers, strong and firm. The contained loculi vary in size from that of a grain of sand to that of a pea; they are round or oval; occasionally adjacent ones communicate together by solution of the interposed wall. The jelly-like substance in their cavities is greenish-yellow, semi-transparent, and clammy; "it yields no gelatine on boiling, but seems to consist of a peculiar substance, identical with that naturally occurring in the cavities of the thyroid, and in some cysts. Cells, nucleated and non-nucleated, caudate and fusiform, nuclei, and elementary granules, occur in this substance, and, under circumstances probably connected with softening changes, granule-cells, and fat molecules. Endogenous production of the cells within parent cells is sometimes observed. Colloid may present itself as a distinct solitary tumour," or may infiltrate the tissues which it infects, when it occurs on serous membranes; there are often small scattered nodules of the growth in the vicinity of the larger.

Fig. 39.



Colloid cancer of a lymphatic gland.

Several varieties of colloid have been described by some pathologists; but there does not appear to be more than one type of structure to which the term colloid cancer is applicable. This is a structure agreeing in its general type with medullary cancer, but distinguished by the gelatinous character and hypertrophy of its cells. These cells have, in fact, undergone a "colloid" metamorphosis, and the whole growth resembles a medullary cancer which has undergone this change; though it is by no means the case that each specimen of colloid cancer has passed through the stage of medullary. The other growths formerly described as colloid are either myxomata, or else some other form of tumour which has undergone a hyaline or colloid metamorphosis.

The favourite *habitat* of colloid are the stomach and omentum; it also occurs in the ovaries, the bones, the kidneys, the uterus, and the spleen, more rarely in the lungs,[*] and, as a secondary growth, in lymphatic glands. Its growth is often rapid, and it may attain a very large size, exceeding that of a cocoa-nut. The contents of the alveoli are sometimes of pearly aspect, probably from the presence of cholesteatomatous matter. Colloid may exist combined with scirrhus, and also with encephaloid; in the latter case, the superficial loculi become occupied by soft encephaloid matter.

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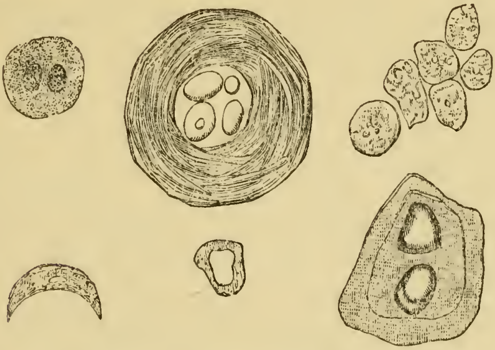
[*] Bristowe. "Trans. Path. Soc.," xix. 228 (1868).

Clinical character.—The malignant character is less marked in colloid than in the other kinds of cancer; it does not induce such marked cachexia, does not so often reproduce itself in distant parts, or contaminate the lymphatic glands, and is less prone to softening and decay, or to inflammation and saniation. As, however, it has a great tendency to infiltrate neighbouring parts, and, in well-marked cases, affects the neighbouring lymph glands, and even reproduces itself in distant organs, there can be no doubt of the propriety of the term colloid *cancer*. The peculiar degeneration which the cells undergo is perhaps the reason why they have a slighter tendency to reproduce themselves in distant parts.

EPITHELIAL CANCER; CANCROID; EPITHELIOMA.

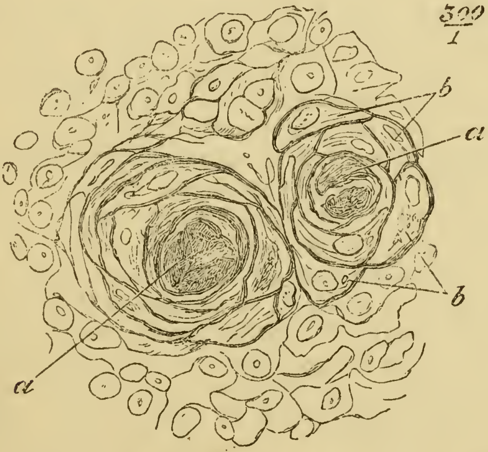
Epithelial cancer seems only recently to have been admitted among the varieties of this disease; it is not mentioned by Dr. Walshe in his elaborate work, and yet its cancerous nature in many cases is unequivocal and strongly marked. It occurs as a primary growth almost solely on tegumentary or mucous surfaces, or in secreting glands; the lips and cheeks are among the parts most commonly affected by it. Rokitansky and Förster mention having observed it in the liver, and Sir J. Paget has recorded two cases of its occurring primarily in lymphatic glands. On mucous surfaces it appears as a cauliflower-like growth, of a more or less red tint from vascular injection, of various degrees of consistency, and easily separated into parts by pressure. On the general tegument, its appearance is most often that of a low, tolerably well-defined tumour, of hard feel, having an irregular nodulated surface, covered with minute watery papillæ: when ulceration and softening take place, the surface becomes injected, a watery and serous discharge is poured out, and gradual destruction of the part proceeds. In structure, these tumours essentially consist of an alteration of the integument, the corium and subcutaneous areolar tissue being converted into morbid growth, the papillæ greatly hypertrophied, as well as the epithelium resting upon them. The epithelium is also found dipping down into the deep parts and infiltrating the co-subcutaneous connective tissue as well as the deeper layers of the skin. In the last specimen we examined, which was from the lower lip, a vertical section displayed an external whitish layer, about one-third inch thick, marked by vertical striæ, and resting upon some areolar tissue, fat, and muscle. Its surface showed but slight traces of subdivision. Its extent in depth appeared to be most accurately limited by the lower margin of the whitish striated layer, but upon examination the areolar tissue immediately subjacent, and for some depth, was found thoroughly infiltrated with nuclei and granular matter. Sections of the altered integument showed papillary elevations, completely overwhelmed and blended together by an enormous growth of scaly epithelium, which in some parts showed a tendency to fatty change, and here and there the capsulating arrangement mentioned below. If glands exist in the part affected, their epithelium may also accumulate within their canals, and thus add to the size of the tumour. One peculiarity is very commonly observable in the arrangement of the cells of epithelial cancer, which does not seem to occur in other growths; this is, that here and there the scaly particles are arranged in lamellæ

Fig. 40.



Epithelial cancer. Four of the loculi are shown. and some of the flattened cells, one of which is curved, having probably been arranged round a loculus.

Fig. 41.



Two birds' nest bodies from epithelioma. *a*. Amorphous or degenerated matter in the centre of the mass; *b*, large epithelial cells arranged concentrically. (From an original preparation.)

around a central circular space, which contains amorphous colloid matter or degenerated cells. These peculiar structures are called "epithelial pearls," "*globes épidermiques*," or "bird's nest bodies." (Figs. 40 and 41.)

Clinical character.—The malignant character of epithelial cancer is manifested in its extending from the superficial textures first involved to the deeper seated, even to the bones; the laryngeal cartilages have often been involved by it. It seems, however, to have less tendency to contaminate the lymphatic glands, and the system generally, than other varieties of cancer; though Sir J. Paget observed some extension to the lymphatic glands in about half his cases. Instances of the occurrence of new growths in distant parts of the body are not unknown, though rare. In some of these cases there has been supposed to be a mechanical transference of germs.*

Cholesteatoma.—A peculiar form of epithelial tumour known by this name, appears to be nearly allied to epithelioma. It contains flat epidermic cells, intermixed with cholesterine crystals. It is extremely rare, and specimens have been probably often confounded with fatty cysts.

Tubular or Cylindrical Epithelioma.—The tumours above described are formed of epithelial cells resembling the flat, squamous, or pavement epithelium; but there are other cancers in which the epithelium was of the cylindrical variety. Tumours of this kind always grow on mucous surfaces, which are covered with cylindrical epithelium, such as the interior of the stomach and intestines. They are soft, present on section a milky juice, and often have a considerable resemblance to medullary cancer; and like malignant growths they infect the neighbouring lymph glands, and occasionally give rise to secondary growths in the liver and lungs. The anatomical characters of this form of cancer are a well-marked stroma and cylindrical epithelial cells arranged in its alveoli, perpendicular to the walls, as in an epithelial surface. Some examples of villous cancer, or "cauliflower excrescence," have this structure.† This form of epithelioma is related to the glandular tumours, as the common form is to innocent epidermic growths.

Epithelial cancer, in general, is a disease of middle or advanced life, being almost unknown under the age of thirty. It is more common in men than women, and often appears to owe its origin to some local irritation, as that of soot in the case of chimney sweeps, who are especially liable to epithelioma of the scrotum.

Cystic Cancer, or Cysto-Carcinoma.—A cystic growth sometimes occurs in combination with one or other of the species of cancer, chiefly with encephaloid: the cysts may be simple or compound. These cysto-carcinomatous growths arise in various ways: either a production of cancer tissue takes in the wall or the cavity of a pre-existing cyst; or cysts are formed by dilatation of natural cavities (as glandular acini) contained in the cancerous growth; or else some softening or colloid transformation of cells causes the production of cysts from the cancerous elements themselves.

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* Moxon. "Trans. Path. Soc.," xx. 28. The malignancy of epithelioma is greatly influenced by physical conditions, being more pronounced in proportion to the moisture, vascularity, and amount of movement in the part. Thus it is far more likely to affect the glands and other parts when occurring on the lips, tongue, genitals, &c., than when growing from the skin of the cheeks or limbs. See Arnott "on Cancer." London, 1872. p. 70.

† See Hutchinson: "Trans. Path. Soc.," viii. p. 254. Cornil and Ranvier: "Manuel l'Histologie Pathologique Générale," p. 280.

Villous Cancer.—Rokitansky describes a variety of cancer, which he calls *villous*, from its consisting of a kind of delicate fibrous stalk branching at its end into villous processes, with somewhat bulbous terminations. They grow on the internal surface of natural cavities, and are extremely vascular. Hæmorrhage often takes place spontaneously from them, and is easily excited by the slightest lesion. It is probable that many innocent tumours, having the same form, have been confounded under this name; but many cases are undoubtedly cancerous. They more commonly belong to the epithelial type of cancer, and are furnished with cylindrical epithelium, but in other cases true encephaloid structure seems to form their basis. They occur in the bladder, in the uterus and vagina, and on the mucous surface of the stomach and intestines. Some such growths have been described as cauliflower excrescences.

GENERAL REMARKS ON CANCER.

There may be, probably, other varities of cancerous tumours, or to put it otherwise, tumours possessing more or less of cancerousness; but we have sketched the outline of the principal forms that are usually met with, and we feel convinced that it is far more important for the student and the practitioner to contemplate steadily the great characteristics of malignant disease, than to load his memory with details of the incidental and trivial. Partly on this account we have not attempted to give any very minute descriptions of the structure of cancerous tumours, for our own examinations have most thoroughly convinced us of the non-existence of any special structural character, absolutely and in all cases distinctive of malignant growths. If a tumour present the characters given above as distinctive of malignancy, it tells its own tale, and anatomical investigation is not necessary to determine this point. But it may happen that it is in so early a stage as not to show these characters, and yet be certain to develop them sooner or later. In these cases anatomical structure is of great value for prognosis, though even then the utmost that can be hoped for is to refer the tumour to its right place in classification, and thus gain a key to the information which has been collected with respect to the usual history and progress of such growths.

Anatomical Characters of Cancer.—What may be said relative to the distinguishing of cancerous from other tumours, by their mere physical characters, and not by their living actions, amounts to this:—If a tumour, on being incised and compressed, yield a whitish, milky juice (the so-called "*suc cancereux*"), it is probably cancerous; we have, however, failed to obtain this sign from actual encephaloid. If the cell-growth of a tumour is what may be called exceedingly *multiform*, *i.e.*, one particle unlike another, the field of view being filled with utter varieties of shape and construction, there arises a strong presumption that the structure is cancerous. If a tumour consist of an abundant cell-growth lying in the alveoli of a fibrous stroma of slight consistence, it so far bears a close resemblance to encephaloid. There are, however, scirrhous tumours (of the breast) in which the cellular elements occupy a very subordinate position, but the alveolar structure will even here be an important criterion. An *epithelial* character of the cells, that is, their being flat, angular from mutual pressure, and closely packed without intercellular substance, has been thought by recent observers, especially in Germany, to be an important distinction of cancer as contrasted with sarcoma, and the absence of intercellular substance is also a character of some weight.

The above characters will serve in the great majority of cases to distinguish anatomically the group of cancers, a group, as we have said, by no means co-extensive with malignant tumours, although the most certainly malignant.

The characters of sarcomatous tumours, which stand next in order of malignancy, we need not recapitulate, except to insist upon the importance of the presence of an intercellular substance, regarded as a positive character, though it may be difficult to see except in a hardened specimen. If a tumour can be satisfactorily referred to this group, we know that it is extremely likely to recur, and may very probably be truly malignant. If finally, we find the tumour to possess the characters of some simple tissue of the body, we know that recurrence, or any form of malignancy, is as exceptional as the opposite qualities are exceptional in the case of the typical cancers.

Scale of Malignancy.—In concluding these general remarks, we may state, we think, the following position with some confidence, viz., that, starting from encephaloid as the representative of cancer *par excellence*, we find the malignant character gradually declining as we pass through a series of formations, such as we have above described, until we come to those of whose innocent nature there is no question. The exact limit, we believe, at which malignancy is lost cannot be distinguished by any clear line, any more than in natural history we can separate absolutely animals from plants, or in chemistry we can make a positive distinction between the metals and the non-metallic elements. Such a scale we have attempted to represent in the following table:—

SCALE OF MALIGNANCY.

Malignant.
Encephaloid.
Scirrhus.
Round-celled or Medullary Sarcoma.
Colloid Cancer.
Epithelioma.
Osteoid.
Spindle-celled Sarcoma (Recurrent Fibroid).
Glioma.
Myeloid Sarcoma.
Myxoma.
Enchondroma.
Fibroma.
Simple Histioid Tumours generally.
Innocent.

Vessels and Nerves of Cancer.—The vessels of cancer for the most part, we believe, are of the ordinary kind, derived from those of the natural tissues by the process of extension or growth: they have generally very thin and simple walls, so as to resemble very large capillaries rather than arteries and veins. Lymphatic vessels have been traced in cancerous tumours, accompanying the blood vessels. They doubtless communicate with the lymphatic vessels of the part affected. No new formation of nerves appears to take place, but the nervous filaments traversing or distributed to the part which is the seat of the cancerous growth, are often involved in the mass, and becoming injuriously pressed on, or otherwise injured, occasion the most frightful pains. There is scarce anything accurately determined respecting the chemical composition of cancer. Encephaloid is said to consist chiefly, if not entirely, of albuminous matter; scirrhus to contain gelatine also, while colloid jelly seems to be a principle quite *sui generis.* Possibly there may be some special cancerous virus, as there is a variolous and syphilitic, but as yet chemistry knows nothing of it, and we only infer its existence from the effects it produces.

Origin of Cancerous Growths.—The *origin* of cancer is a subject of the deepest interest, but the first and most important step of the process is entirely concealed from us. Mr. Simon views a cancer as "substantially a new and excretory organ," a growth which arises for the purpose of eliminating from the system an unhealthy matter which is generated within it. Such a function, however, is surely not fulfilled by all instances, even of encephaloid, and still less of the other species. It rather seems that we should recognize in cancer a grave alteration of the normal formative powers; those real, but occult influences which determine that—here bone, and there muscle, and there nerve shall be produced. We know something of the disturbances of nutrition occasioned by unhealthy conditions of the blood *quoad* its chemical composition; we know something of the origin of rheumatism, and gout, and syphilis, and of the effects they produce; but they are very different from the phenomena of new formations. Surely the arising of a fibrous tumour, an enchondroma, or a cancer, implies a very different kind of action to that which is observed in any blood disease. We certainly believe the blood to be affected, probably in its chemical composition, as well as, and most importantly, in its vital endowments, but we do not think it is the only seat of primary alteration. Were it so, how would it be possible to account for the constant preference manifested by scirrhus, and by other tumours, for particular sites? We conceive then, that in the case of cancer, the blood and the general system, but especially some particular part, having suffered some unknown deterioration or perversion of their vital power, a formative process is set up in the specially weakened part (perhaps in consequence of a blow or other injury), causing the development of cell and fibre structure, which soon constitutes a new growth, endowed with powers of assimilation and vegetation to an almost indefinite extent. The tendency to cancerous disease, that is to say, the deterioration of the blood, and of the assimilative powers, may exist for a long time before it expresses itself in the tangible reality of a tumour; but when this is formed it becomes an engine for multiplication of similar tumours, and intensification of the cancerous diathesis. It is not possible to trace the process further back than to the tissues themselves. In the vast majority of instances the growth manifestly originates within or in the interstices of textural elements; in some few it has been found in the coats of the vessels, but in none has it been certainly proved to have originated in the blood. Cancerous growths have, indeed, been seen within the veins, but this has been the result of perforation of their walls by formations external to them. Development proceeds in rudimentary cancerous tissue just as it would do in healthy; nuclei and fibres seem to arise in the same way; and the view taken of the pathological process must depend upon the light in which the physiological processes of growth and development are regarded.

Theories of the development of New Growths.—According to one theory the formation of new elements, whether in physiological or pathological circumstances, has been regarded as a production *de novo*, or as the transformation of an amorphous substance into formed elements, much as a saline solution transforms itself into crystals. This may be called the *blastema* theory, or the theory of free cell development. On this theory the formation of a new growth begins with the exudation (from the vessels) of a certain quantity of blastema, which proceeds to develop cell and fibre-structure. In the case of morbid growths, such as cancer, the special character or morbidity of the growth depends upon the blood and general system, but especially on the particular part in which the growth takes place having suffered some unknown deterioration or perversion of its vital power. In opposition to this view has been elaborated the theory of cellular development, founded in great measure upon the facts of embryology, and purporting to unite in one view the formative processes both of vascular and non-vascular parts. According to this view every cell is the progeny of some other cell—*omnis cellula e cellulâ*—and just as in the formation of an embryonic limb the generations of tissue-elements may be traced back to one original ancestor, which first began to multiply by division or endogenous cell development (Goodsir, Remak), so in the case of pathological new growths, their component cells may be traced back to pre-existing tissue-cells, of which they are the lineal descendants. What, in the case of new growths, determines the character which they assume is as little explained on this as on the other view; we do not even know whether it is a general cause affecting the whole body, or a special one affecting the part. All that is asserted is that the cause affects the original elements of the part as a special stimulus, causing them to take on some special kind of development. The evidences of this development are derived, *firstly*, in a general way from the fact that a new growth always takes its rise from some special tissue in an organ, *e.g.*, from fibrous structures, or from epithelium, and that this mother-tissue, or matrix, has great influence on its type of organization; *secondly*, from the signs of multiplication, either by division, or more rarely by endogenous proliferation, which may be traced in the elements of the surrounding parts. In many cases transitional forms are met with which connect the original tissue-cells with the elements of the new growth. Thus, if we examine the peripheral portions of a cancer of the breast, we find the adipose tissue more or less infiltrated, as it is said, with cancerous growth. This infiltration consists in the gradual transformation of the adipose connective tissue into cancer, and is effected by the production from the connective tissue corpuscles of nuclei and elements resembling the cells of the cancer, the interstices disappearing by the pressure of the cell growth. Here several stages may be seen, such as (1) a connective tissue corpuscle, containing several nuclei; (2) another containing several cells in place of the nuclei; (3) another much enlarged, and containing many cells, so as to resemble precisely an alveolus of the cancer itself. In this case it is concluded that the process consists essentially in the conversion of the connective

Fig. 42.

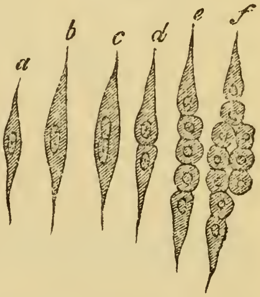
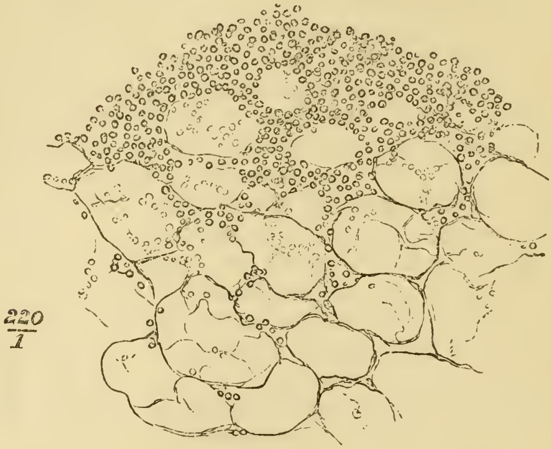


Diagram of the proliferation of connective tissue. *a.* Simple connective tissue corpuscle. *b.* The same enlarged or hypertrophied. *c.* Division of the nucleus. *d.* Division of the cell. *e.* Further division of the cell and formation of round granulation cells. *f.* Continued proliferation.

(After Virchow.)

tissue corpuscles into cancerous alveoli, an explanation which if the observations on which it is founded are correct, accounts for the formation of by far the greater part of the tumour, and by analogy must be extended to its original formation.* In some cases we find the surrounding elements converted, not into special cells like those of the cancer, but into groups of nuclei or small corpuscles; and these becoming continuous, ultimately form a mass of what has been called "indifferent granulation material," which, it is thought, becomes transformed into the cancerous tissue. It is not certain that this is always the case, or that the production of cancer always passes through this stage. Both this process (represented in Fig. 43) and the more direct tranformation into cancerous elements may be seen simultaneously. That now spoken of has nothing specific about it, but is the same for all kinds of new growths. The plan of growth in different organs may be various, but the essential point is that the new elements are formed by division and multiplication of those already existing. Another way in which new elements are produced is by the migration of leucocytes from the vessels.

Fig. 43.



Connective tissue from the neighbourhood of a tumour, showing nuclear proliferation or infiltration with small cells, which leads to the formation of "indifferent granulation material."

Relation to Epithelium. — It was at first imagined that one kind of tissue only, namely, connective tissue, was able to become the matrix of new growths, and there is no doubt that it is so more frequently than any other. One of the most important contributions to science made by Virchow consists in his having demonstrated the existence of this tissue, and the possibility of new growths arising from it in organs such as muscle, nerve centres, &c., formerly supposed to be destitute of it. But more recent inquiries have shown that other tissues also, more especially epithelium (both that of mucous and glandular structures, and the serous epithelium, or "endothelium" which lines the serous cavities and lymphatic channels) and the nuclei of blood-vessels, may serve the same purpose; and it is not certain that even highly-specialized elements, such as liver cells, may not sometimes give rise to new elements. In the case of cancer especially the question has given rise to much discussion. Virchow first laid down the general principle that cancers, excluding epithelioma, were always developed from the connective tissue, or some one of the less specialized tissues; but of late years it has been shown by Waldeyer* and others that some forms of cancer arise from glandular or epithelial structures. It is, however, admitted that in these cases the growth soon spreads to the connective tissue, and hence the infection of the latter may still be the real distinction of cancer from those growths which are simple glandular hypertrophies, and cancer would then be, as defined by Billroth,† a primarily epithelial growth, with marked and intense infection of the connective tissue. This local infection is in fact the clearest indication of truly cancerous growth. Since this question is certainly not at present completely set at rest, we have not thought it necessary, for the purposes of this manual, to distinguish the epithelial and connective tissue cancers further than to separate those which are at first, and remain epithelial, under the name of *epithelioma*.

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* It is not necessary here to consider the possibility of such a cancer having originated in the glandular epithelium, since even in that case it would, by affecting the connective tissue, have produced the phenomenon here described.

Relation to Lymphatics.—A second very important point in the development of cancer is its relation to the lymphatic system. It has been shown by MM. Cornil and Ranvier, and also by Dr. Köster for epithelioma, that the alveoli of cancer communicate freely with the lymphatic vessels of neighbouring parts. This fact explains in a very striking manner the direct and almost constant infection of the lymphatic glands, which is so characteristic of cancer. It is, moreover, by no means inconsistent with the close connection of cancer and connective tissue, if we accept the views of His and others, that the connective tissue forms a system of plasmatic channels, in close connection with the lymphatic system. In the case of glandular cancers and epithelioma the long tubular prolongations filled with cells, which are found to extend into the neighbouring tissue, seem also to be casts of lymphatic vessels. Although these facts make it clear how some infecting material passes from the tumour to the nearest lymphatic glands, it is by no means certain what that material is.

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* Waldeyer. Virchow's "Archiv," xli. p. 470.

† Billroth. "Chirurgische Pathologie und Therapie." Third edition, 1868, p. 697.

Whether the actual transport of cells is necessary, or whether some smaller germs or even some fluid substance may convey the stimulus which leads to production of cancerous structure, is at present quite undetermined.

GROWTH AND DEGENERATIONS OF CANCER.

The growth of a cancerous tumour will be more rapid, in proportion as its structure is mainly composed of cells, and contains but little fibre, and is also so situated that it has room to expand freely. Encephaloid, in which cell-structure always predominates, is notoriously of most rapid growth, but even its progress is comparatively slow while it is confined within unyielding walls, as in the globe of the eye. Laennec supposed that all cancers were originally hard, and that in the process of growth they gradually became softer. This is not so; cancers may be quite soft at their commencement; but still they do generally appear to diminish in consistence as they advance in age. Tumours, however, of the same date may differ very greatly in consistence. Inflammation may affect cancerous tumours; it is commonly excited by their exposure to the air after they have made their way through the covering parts. It powerfully accelerates softening and decay. Suppuration may take place as the result of inflammation, but the pus is an ill-formed sanious product, mingled with detritus. Cancerous formations sometimes mortify spontaneously, sometimes in consequence of inflammation; in the first case a cure has been known to take place, and to attain this artificially is the object of various escharotic applications. It is evident that it is only in instances of cancer whose powers of vegetation are feeble and sluggish that such a proceeding can be successful. There are other changes which cancer occasionally undergoes, and which, it has sometimes been thought, may result in a cure. One is fatty degeneration. It is this change, in an early stage, which constitutes *Carcinoma reticulare;* it occurs with formation of granule cells, or independently of them. It may attain such a degree as to cause liquefaction and the formation of a cavity. The other change is a shrinking and contraction, a kind of drying up of the cancerous growth, sometimes attended with deposition of calcareous matter, analogous to the cretification of tubercle. These atrophic processes produce the appearance of "umbilication" in mammary and hepatic cancers; and the well-known retraction of the nipple in the former. Another degeneration is the so-called "colloid metamorphosis," in which the structure becomes homogeneous, translucent, and gelatinous. This, which affects specially encephaloid, constitutes a transition to colloid cancer, properly so called.

Primary and Secondary Growths.—The term primary is applied to cancerous growths, originating for the first time in the system; secondary, to those that are in some way derived by dissemination from the primary. We have already alluded to one mode by which secondary cancers are established, viz.:—that through the medium of lymphatics; and we have shown how it is explained by the close connection of both epithelial and connective tissue cancers with the lymphatic system. It is the commonest mode of transmission, especially in the case of true cancers, while in the class of sarcomata, even when they are highly malignant, it is less frequent, though not unknown. In the same way the blood-vessels may be the channel of infection, and the situation of the secondary growths often clearly shows this to be the case. The nidus is most often the next capillary plexus at which the blood arrives; thus, cancer of the breast occasions cancerous tumours in the lungs in almost all cases in which it also affects the liver, but it often produces cancer in the lungs without any occurring in the liver; cancer originating in the stomach occasions similar disease in the liver to which its veins proceed, before it produces any in the lungs. This method of transmission is the rule in malignant sarcomatous tumours, where the lymph-glands are sometimes remarkably unaffected.

Distribution by the Vessels.—In most of these cases of infection by means of blood or lymph-vessels, there is no possibility of tracing by what particles, whether solid or liquid, the secondary growths are set up; but there are instances where it is clear that the primary morbid growth has made its way into the vessels, and either crept along them till it has reached the next capillary plexus, or else allowed of the detachment of solid masses which have been carried along with the circulation till arrested in the capillaries, after the manner of an embolus. These facts have been observed both in cancerous and sarcomatous tumours, and in a few cases of enchondroma. A similar mechanical transport may, however, take place in other ways than by the circulating fluids. Thus the distribution of cancer from the kidneys to the bladder may take place along the ureters; from the larynx to the substance of the lungs, along the bronchial tubes; and in one remarkable case, a transplantation of cancer was observed from the upper to the lower part of the spinal canal, without any intermediate growth. Small cancerous growths have been observed in the track of an operation-wound for the removal of abdominal cancer, and when a tumour (cancer or sarcoma) softens and bursts into the peritoneum, we find an abundant crop of new growths, which spring up, as if from seed. In such cases, the germs or solid masses take root, so to speak, in their new soil, and begin an independent existence.

Degrees of Infective Properties.—It is generally true that the greater the proportion of fibre in a tumour, the less is its malignancy; especially intending thereby its tendency to contaminate the system, and to destructive ulceration. On the other hand, the more it abounds in cell growth, and in fluid blastema, the greater is its malignant capacity. M. Simon says, "in proportion as the blastema has suffered itself to undergo a fibrous transformation, in such measure I cease to recognise that which is distinctively cancerous and malignant." Believing this to be in a great part true, we must still remember that the abolition of a truly scirrhous growth may be followed by the development of encephaloid; the inactive fibroid structure is still the expression of the comparatively quiescent constitutional infirmity, which may at any time be roused to its more severe manifestations. Virchow has pointed out that malignant properties are manifested chiefly in two directions—in the infection or infiltration of the neighbouring tissues, on the one hand, and in the general infection of the system with production of tumours in distant parts, on the other hand; and that sometimes one of these forms predominates, sometimes the other. The difference may depend partly upon a certain energy or virulence of the parenchymatous juices produced in the tumour, but is more closely connected with the vascular arrangements; local infection being characteristic of slightly vascular growths, general infection of those which are abundantly permeated by blood and lymph; a distinction which, as a general, though not an absolute rule, seems to be very valuable.

Pathologists are generally agreed in attaching little credit to the results of the experiments which have been made relative to the production of cancer by inoculation, or injection of the cancerous matter. Some few are said to have succeeded, but the great majority have failed; in this there is nothing surprising; and it has hence been concluded that the non-production of cancer by inoculation proves conclusively "the absolute necessity of constitutional predisposition" for the development of the disease. A healthy system will resist and overcome by an assimilative force the cancerous poison, just as it will in the case of a dog that of pus, while a weaker system might be infected by it, as the rabbit is by the injection of pus. What has been called the *metastasis* of cancerous tumours is said to have been occasionally observed, but the cases as yet recorded are open to doubt. (*v.* a case quoted by Dr. Walshe, p. 110, from Recamier.)

Local Origin of Cancer.—It might be reasonably conceived that the structure of the locality in which cancerous growth arises would influence the kind of tumour that was therein developed, and such does appear to be the case, at least to some extent. Thus scirrhus is far more common than encephaloid in the female breast, which abounds in fibrous tissue; and encephaloid is most frequent in the liver, which contains little of it, and chiefly consists of cells; the muscular walls of the stomach are commonly also affected by scirrhus, as well as those of the uterus, and both these consist mainly of fibres. On the other head there are exceptions in the case of the lungs, and of the meninges which are most often attacked by encephaloid. The species of cancer termed epithelial, seems certainly to be determined by the peculiarity of its site, as it seems almost invariably to be developed on free cell-bearing surfaces.

Inheritance of Cancer.—There is much probability in the common opinion that cancer is an hereditary disease. Dr. Walshe acknowledges his belief in the disease having thus originated in some cases which he witnessed; and, indeed, most persons might adduce confirmatory testimony. Still actual statistical proof has not yet been afforded. Cancer is, on the whole, a disease of advancing age; it does indeed exist, occasionally, at every period of life; both scirrhus and encephaloid have been observed in the fœtus, and encephaloid is not uncommonly seen in infants and children of tender years; but the researches of Dr. Walshe show that the mortality from cancer "goes on steadily increasing with each succeeding decade until the eightieth year;" so that taking the mean of both sexes, it attains its maximum between the ages of seventy and eighty. In males the ratio of increase is more uniform than in females; in them there is a great and rapid increase of mortality between the ages of thirty and fifty, which "lends support to the current belief respecting the connection of the development of uterine and mammary cancer with declining activity and cessation of the genital functions." The influence of sex upon the development of cancer is very striking; Dr. Walshe's tables show that in six years an absolute number of deaths from this disease, in males, was only 3,495, compared with 10,146 in females, although the mean rate of mortality in the male exceeds that in the female. A sanguinous temperament is considered by several who have paid attention to the question to predispose in some measure to cancer. More powerful are the effects of mental distress, and of the refining, but enervating influences of civilized life; at least, such are the conclusions to which the evidence that can be obtained at present seems to point. A fact of a rather opposite import which Dr. Walshe establishes, is that a town life has no greater influence than a country one, in promoting the development of cancer; in fact, a greater number die of cancer in the country than in the towns; this applies to the mean of both sexes, but taking the females separately, the mortality from cancer among them is greater in the town than in the country.

Causes of Cancer.—An injury to a part (the observation is most common in the case of mammary cancer) seems to be often the exciting cause of cancerous development, and it is probable enough, and accordant with the analogy of growth, that a weakened part should offer the least resistance to the localization of constitutional disease. Epithelioma certainly often arises in a wounded or irritated spot. However, the immunity from cancer observed in the Parisian prostitutes, and the result obtained by Andral, with regard to cerebral carcinoma, as following injury, show that but little is to be attributed to this as an exciting cause. Habitual irritation of the stomach by alcoholic liquors has no effect in inducing carcinomatous disease of this organ, though it may have sometimes produced a state of simple induration, which has been mistaken for scirrhus. It is, however, not to be denied, that irritation may in some cases determine the formation of a cancer; as, for instance, in the scrotal cancer of chimney-sweeps, which certainly seems to be called into existence from a latent predisposition by the irritation of soot.

Results of Operations.—In concluding this subject, we may offer a few remarks with reference to the effects of removing cancerous tumours by operation. In the first place, it is quite clear that the disease is manifestly constitutional, and that no sound, real cure can be expected from merely removing its external development. Secondly, it is matter of experience, that in not a few instances surgical interference with one tumour has provoked the speedy appearance of several others. Thirdly, any attempt at removal is useless; nay, may be absolutely injurious, unless every particle of cancerous structure is taken away. Fourthly, epithelial cancers seem least prone to return after removal; encephaloid invariably does, and mostly with great rapidity; scirrhus may be checked in its progress, but its return can very rarely be prevented. The check which may be given by operation to the progress of cancer depends on the circumstance before stated, that a tumour, once formed, becomes an instrument for the multiplication of similar tumours and intensification of the diathesis. It must require a combination of favourable circumstances, or a great intensity of the diathesis, to ensure the development of effused blastema into an heterologous growth, but when this has taken place, then the very growth and vital actions of the structure will constantly generate fresh supplies of cancerous blastema, and thus promote the formation of secondary cancers. The destruction, therefore, of the growth, which thus reacts so evilly upon the system, may be reasonably expected, *if it do not aggravate*, to delay the cause of the disease. But the misfortune is, that, as above stated, it does sometimes aggravate, and that fearfully, a previously indolent cancerous diathesis. Dr. Walshe says, "excision of a tumour seems to awaken a dormant force, cancers spring up in all directions, and enlarge with a power of vegetation almost incredible." Why this should happen we do not know, but we may conjecture that when the original diathesis is slight, the formation of a tumour may tend in some degree to localize it, and leave the system in a somewhat healthier state, provided the tumour itself be chiefly fibrous, and produce but a small amount of blastema. The removal of the indolent tumour may be analogous to the cure of fistula in ano in a person of phthisical tendency. The two principles referred to of the cancerous tumour, in one case acting as a cause, increasing the force of the disease, and in another retarding it, are not contradictory, though opposite; they will prevail in different degrees in different instances, according to the kind of tumour, and other circumstances.

CHAPTER VI.

TUBERCLE AND ALLIED PRODUCTS.

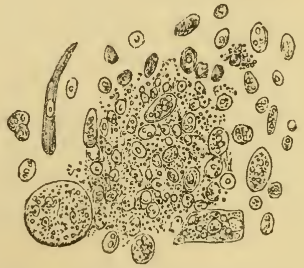
We have here to consider certain morbid products which have been sometimes classified as a peculiar kind of exudation, sometimes as new growths, sometimes as inflammatory products. They occupy as we believe an intermediate place between the two latter. They include—(1) Tubercle; (2) Products of constitutional syphilis; (3) Lupus; (4) Morbid products of glanders, and, perhaps, other specific diseases.

TUBERCLE.

The term tubercle has been applied to objects so different that when the extremes are compared it will seem surprising that they should have ever been denoted by the same term. There are, however, two principal classes of these objects, distinguished as *grey* and *yellow* tubercle.

Grey Miliary Tubercle.—The former, sometimes called grey granulations, are about the average size of a millet-seed, but vary from $\frac{1}{12}$in. to $\frac{1}{250}$in. in diameter, roundish, resisting under pressure, of a greyish, semi-transparent aspect. The microscope shows them to consist of nuclei, small simple cells, and some larger cells with many nuclei. The existence of free nuclei has been denied by some writers, but we often find very small bodies in which no method of treatment exhibits a nucleus; and which therefore seem to be nuclei themselves. The predominant elements are, undoubtedly, small cells from about $\frac{1}{6000}$ to $\frac{1}{3000}$in. in diameter, consisting

Fig. 44.

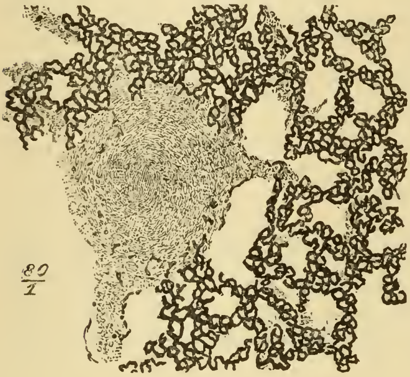


Elements from grey tubercle; miliary granulation.

of a relatively large nucleus, surrounded by a small mass of protoplasm. They sometimes approach both in size and appearance the white cells of blood or lymph. Large polynucleated cells of irregular shape, or "mother-cells," like the "myeloid" cells of bone are also often met with, but they are not constant. These elements are, according to some observers, imbedded in a homogeneous basic substance; but this is, in some instances, very scanty, and is extremely difficult to demonstrate; according to others, the cells are contained in an extremely fine reticulation, like that of a lymphatic gland, a kind of structure which has been called *adenoid*. We believe that sometimes a tubercle does develop into such a structure, but that in general it stops short of this degree of development.

The arrangement of these elements in the miliary tubercle is very curious and characteristic. The peripheral portions consist of distinctly formed cells, but towards the centre these become smaller and less definite in outline, and the central part of the tubercle is composed of amorphous or granular matter, which is sometimes decidedly fatty, but has sometimes been described as horny. Since the central portions are the oldest, we conclude that this amorphous condition is produced by the atrophy and decay of a structure previously composed of formed elements. The fact that when the structure has reached a certain size its elements begin to decay, is very characteristic of tubercle, and is probably connected with the fact that this growth, when it has reached this stage, contains no permeable vessels. In Fig. 45, which represents a minute miliary tubercle from the lung, it will be seen that the very complete injection of the capillary network does not enter the tubercle. This does not show that no new vessels formed, but that they very early became obliterated. A tubercle thus seems to be a structure intermediate between the products of inflammation and the new growths properly so-called, being less transitory than the former, less permanent than the latter, and less capable of development. This form of tubercle never occurs solitary, and is generally seen in very large numbers. The disease in which they are produced has been termed tuberculosis. It is to be noticed that we generally speak of *a* grey miliary tubercle, or of *tubercles* in the plural; but it is not certain that the same product may not sometimes occur in a more diffuse form. They are best studied on serous membranes,

FIG. 45.

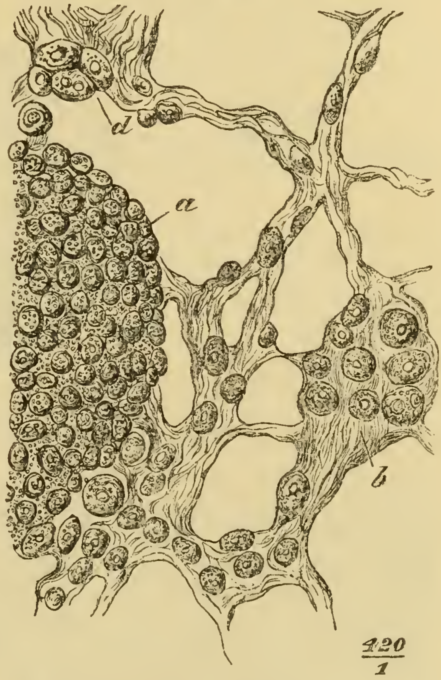


Grey miliary tubercle of the lung, surrounded by healthy lung tissue, the capillaries of which are completely filled by injection from the pulmonary artery. (From an original preparation.)

especially the omentum or the pia mater of the brain; it being difficult to distinguish them in the lungs from other small masses, which owe their form to the anatomical structure of the lungs.

Miliary tubercles are for the most part, though not invariably, formed around vessels, and not so much round capillaries as round the smaller arteries and veins. The origin of the elements composing tubercle is variously explained, and three views must specially be mentioned. In the first place it has been supposed that a homogeneous blastema is first effused in the fluid condition, and becomes organized into a tubercle. Quite lately, Schüppel has contended that the origin of tubercle is free cell-formation from blastema within a vein. In the second place it has been held that the elements of tubercle are derived from the elements of the tissues by proliferation; that is to say, that they are descendants of the connective-tissue nuclei in the walls of arteries, and the nuclei of capillaries; or of the epithelial structures lining the lymphatic perivascular sheaths (of cerebral arteries), and other forms of serous epithelium (see Fig. 46). A third hypothesis is that the original tubercle elements are white cells of the blood which have passed through the vascular wall. It is impossible to discuss these theories here, since the preference given to one over the other must depend upon general principles; the evidence not being complete in any particular case. It is only certain that in the vessels of the pia mater and brain, where the relations of tubercle to vessels can be best studied, the first growth begins in that space between the proper wall of the vessel and its outer sheath, which is sometimes called a lymphatic space.

Fig. 46.



Miliary tubercle of the omentum and changes in surrounding parts.

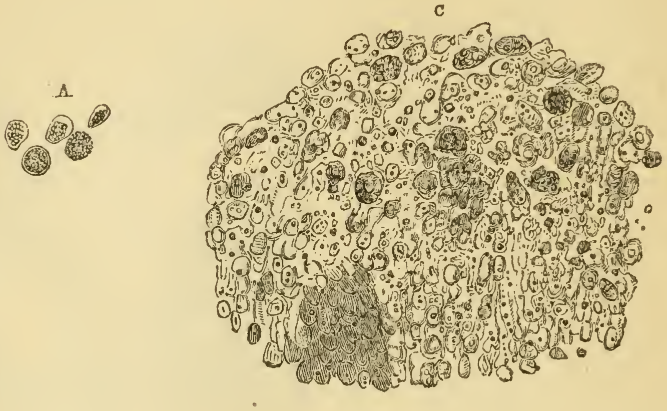
a. Tubercle completely formed. *b*. Cluster of small cells forming an incipient tubercle (mass of adenoid tissue, Sanderson). *d*. Proliferation of serous epithelium.

(From an original preparation.) See "Trans. Path. Soc.," xxi. 198, pl. V.

Finally, it should be stated that Dr. Burdon Sanderson believes all miliary tubercles (at least as produced experimentally) to be enlargements of previously existing masses of adenoid tissue, resembling very minute lymphatic glands, which he has been the first to observe in many organs.*

Caseous Material or Yellow Tubercle.—The substance which has been called yellow tubercle forms masses of varying size, but generally larger than grey tubercle, equalling perhaps a hemp-seed, or a pea, in magnitude; but masses much larger than this occur in the brain, and in lymphatic glands. So little uniformity is there in the size or mode of growth of these masses that it seems more natural to speak of yellow tubercle as a substance, that is, as tuberculous matter, than of *a* yellow tubercle or *tubercles.* This yellow substance has also been called scrofulous matter, and since both these names may give rise to misunderstanding, some writers prefer to speak of it simply as cheesy matter. Whatever view be taken of the origin of the two forms of so-called tubercle, there is an obvious convenience in denoting them by different names; and it therefore seems more convenient to reserve the name tubercle for the grey form, and to call the yellow cheesy masses simply caseous material. It is an opaque substance of a whitish-yellow colour, and of rather brittle consistence. Microscopical examination shows it to consist of amorphous molecules, many fatty granules, and small, shrivelled, angular bodies, preserving a good deal of cell form, which are in reality atrophied and withered lymph or pus-corpuscles. These are the well-known "tubercle corpuscles" of Lebert, at one time thought to be diagnostic of tubercle. (See Fig. 48.)

FIG. 47.



Caseous material or "yellow tubercle."

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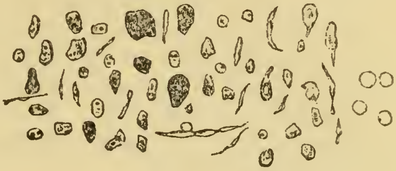
* "Eleventh Report of the Medical Officer of the Privy Council for 1868." London, 1869. Appendix, pp. 91-125.

Origin of Caseous Material.—Scrofulous or caseous material is the result of more than one morbid process; Billroth thus summarizes the modes in which it may be produced: (1) It may be the immediate product of acute cellular infiltration or acute inflammation. This is most clearly seen in rabbits and guinea-pigs, when irritation is experimentally set up under the skin by a seton; this causing not suppuration or ulceration, but the formation of a dry, yellow friable mass. It is not quite impossible (in Billroth's opinion) that a similar process takes place in some scrofulous affections of children. (2) Most commonly some new formations, consisting chiefly of cells, such as hyperplastic lymph glands, tissue infiltrated by chronic inflammation, the central parts of a miliary tubercle or of a tumour, such as cancer or sarcoma, &c., undergo a caseous metamorphosis, that is to say, the vessels become obliterated, the tissue breaks down into molecular substance and dries, while the fluid is absorbed by the surrounding vessels. The same process may take place in tissues which are infiltrated with blood, such as the hæmorrhagic infarctus of the lungs, spleen, or kidneys met with in heart disease; though the more common result is for these masses to be completely absorbed. (3) A caseous mass may also result from the inspissation and degeneration of pus, if an abscess become incapsulated and prevented from discharging its contents; but this mode of origin is not so common as has been supposed. The whole process has received the name of caseation or *tyrosis* (τυρός=cheese); the result is, we may repeat, the production of what is called indifferently yellow tubercle or scrofulous or caseous matter. This caseous or scrofulous material undergoes two metamorphoses of very great importance; one is that of *softening*, the other that of *cretification*. Softening consists in the texture of the mass becoming more lax and moist, with notable increase of size, the change proceeding till it breaks up into a yellowish, diffluent, cheesy mass, which finally becomes a thin, whey-like fluid, of acid reaction, containing minute flocculi. The change seems first to affect the homogeneous basis-substance, which dissolves into a kind of fluid, loaded with pulverulent molecules; the corpuscular elements in consequence of this are set free, and, at the same time, are themselves more or less corroded and dissolved. The softened tubercle thus consists of (1) a fluid loaded with diffused granulous matter; (2) traces of altered nuclei and cells; (3) free oil in the form of various sized drops. It may also contain débris of the tissues. The cretifying change consists in the gradual

Fig. 48.



Wasted cells or so-called tubercle corpuscles. On the right are four blood globules.

deposition and liberation of calcareous particles in the tuberculous mass, together with simultaneous absorption of the animal matter, and consequent decrease in size. It is said by Rokitansky never to take place, except in softening, or softened tubercle; but this is, probably, too absolute an assertion. The cretified tubercle very often remains as a hard, irregular mass, surrounded by indurated tissue, and appears to be insusceptible of further change: sometimes, however, absorption proceeds further, and almost the whole of the deposit is removed. But when this is the case it is probable that absorption had predominated over the deposition of calcareous matter from the first.

CONNECTION OF GREY AND YELLOW TUBERCLE.

If we were to be guided merely by the descriptions just given of the physical characters and development of grey tubercle, and scrofulous or caseous matter, we might wonder that they were called by the same name, or supposed to have any connection with one another. The actual connection between the two is, however, doubtless very close, though there is not yet a perfect agreement as to its nature. In the first place these two products are constantly found in the same body, and appear to be associated with the same diseases; and in the second place they are often found in the same organs, and in such a way that one appears to be either produced by or transformed into the other. With respect to the first point, there are two very different views. It has been thought by some, since the time of Laennec, that the grey miliary tubercle marks the early stage of a disease of which yellow scrofulous masses characterize a later stage; while more recently it has been asserted by German pathologists (Buhl, Niemeyer, and others) that miliary tubercles are a secondary result of the presence in some part of the body, of caseous products. A corresponding difference of opinion prevails with regard to the local connection of the two. Laennec maintained that the grey tubercle or grey granulation was the nascent phase of the yellow, and drew attention to transitional forms between the two. According to more recent views the caseous or softened scrofulous matter causes, by a kind of local infection, the production of miliary tubercles in its neighbourhood.

Change of Grey into Yellow Tubercle.—We may say at once that we have no doubt that the views of Laennec are correct in a certain sense and in some cases; but they are not the whole truth. Doubtless grey miliary tubercles often represent an acute disease, or, perhaps, the early stage of a chronic disease; and if they remain longer do often become replaced by yellow tubercle, or at least yellow tubercle is formed in the same place; only we believe it to be possible to draw anatomically a distinction between tubercle and alterations of surrounding tissues; for we find that the yellow masses are formed not by the transformation of the tubercle alone, but also of the secondary products of inflammation formed round it, so that a mass of yellow tubercle often contains a grey tubercle. This is seen in the lung, where a grey tubercle becomes apparently enlarged by the (inflammatory) products in the neighbouring air-cells. These inflammatory products do not at first differ apparently from those of simple inflammation, but have a peculiar tendency, instead of undergoing simple absorption and removal, and leaving the tissues intact, to break down into amorphous granular material, and involve the tissues in their ruin. This is the *caseous transformation* of tubercle which we believe really to exist, so that, to the sources of caseous masses before enumerated, we ought to add in a prominent place, the products of tubercular inflammations. We shall see, in speaking of the peritoneum, that the tubercle, with its associated products may undergo another kind of transformation and become fibrous. This is, in fact, the general rule with tubercles of serous membranes.

Theory of Secondary Production of Grey Tubercles.—The arguments for the more modern view that miliary tubercles are secondary to caseous masses, require to be stated more at length. They were originally derived from pure observation in the field of clinical and post-mortem examination, but have received a certain confirmation from experiment. Observation first led to the hypothesis that the production of miliary tubercle depended upon the absorption of degenerative inflammatory products, producing rather a local or constitutional infection of the tissues. The following are the chief facts adduced by Buhl, who, with Dietrich, has been the principal defender of this doctrine. (1) The almost constant presence of yellow tubercle or pulmonary cavities in cases of miliary tuberculosis. (2) The yellow tubercle is in these cases not incapsulated, and the vomicæ are not enclosed all round by dense fibroid tissue, and hence absorption is easily possible. (3) The accumulation of miliary tubercles first begins in the neighbourhood of the seat of the primary disease, and they are at first small, soft, and grey. (4) The miliary tuberculosis spreads excentrically, so that the older tubercles which have become altered and yellow, are nearest to the focus, while the smallest or youngest are situated at the greatest distance from it. (5) The disease has clinically the character of an "infective" or so-called zymotic disease.

The theory, as at first stated, implied that, by the caseous metamorphosis of inflammatory products was generated some infective substance or poison, which was the exciting cause of secondary tuberculosis; but it was soon found necessary greatly to enlarge the conception first formed of the antecedent morbid changes; and to include various processes resulting not in caseation but in loss of substance, such as gastric or intestinal ulcers. Instances of such lesions as the probable primary source of tubercle are given by Valentin,* from post-mortem examination, and a case in which a similar conclusion was arrived at from the clinical history is recorded by Murchison.† In some cases acute tuberculosis appears as if it were a primary or idiopathic disease; nevertheless, minute investigation has never, in the editor's experience, failed to detect some results of antecedent inflammation, such as caseous lymphatic glands, cavities in the lungs, or ulceration of mucous surfaces; or even simple inflammation, as pleurisy. It may, however, be fairly objected that the bodies of few persons, such as those who usually die in hospitals, will be free from evidences of bygone inflammation, whatever may have been the disease to which they ultimately succumbed. In one remarkable and probably unique instance of auto-inoculation with caseous matter caused by the opening of a softened "scrofulous" gland direct into a vein, the disease which resulted was not tuberculosis, but pyæmia.‡ The consequences, however, of gradual absorption of the same material might be very different from those of rapid absorption.

It has also been a matter of discussion whether the general infection was due to the passage into the blood of some special chemical substance, or of material particles; and difference of opinion has also arisen as to whether the poison was to be considered as specific, that is, the result of one kind of original disease only, or as the product of ordinary inflammation.

Artificial Production of Tubercle.—Of late years it has been found possible to produce tubercle artificially in animals previously healthy, and the results thus obtained are of great value in modifying our ideas of the nature of the disease. Villemin (1865) first made systematic experiments in this direction. He found that when masses of finely-divided tubercle, yellow or grey, or the products of scrofulous affections, were introduced under the skin of certain animals, especially rabbits and guinea-pigs, the result was, beside a certain amount of local inflammation, that on killing the animals after two or three weeks miliary tubercles were found in the lungs. The same appearances were seen after a somewhat longer time in the intestines and peritoneum. The conclusion drawn by Villemin from these experiments was that tuberculosis must be classed as a specific infective disease, capable of being conveyed by inoculation, like small-pox or syphilis. These experiments have been repeated by very numerous pathologists, and after much discussion the positive results of Villemin have been confirmed, viz., that it is possible by the means he employed to produce miliary granulations in the lungs and other organs, and that these granulations are anatomically identical with miliary tubercle. It was, however, soon found that the same results were attained by the subcutaneous inoculation of many other substances, such as pieces of pneumonic lung, various kinds of pus, putrid muscular tissue; and a further step was taken by Dr. Wilson Fox and Dr. Sanderson, who found that simple mechanical irritation, such as that produced by the introduction of a seton under the skin, might lead in the animals experimented on to the same general infection as the inoculation of "specific" or inflammatory products.* It seemed as if any irritation of the subcutaneous tissue intense enough and of sufficiently long continuance, or any external injury not immediately fatal, might under some circumstances be enough.

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* Virchow's "Archiv.," xliv. p. 296.

† "Trans. Path. Soc.," vol. xx. 1869, p. 174.

‡ "Trans. Path. Soc.," vol. xxii. 1871.

In these experiments it was clear that if the absorption of any morbid poison caused general tuberculosis, it was one generated in, not inoculated, into the wound; and there was, of course, nothing to forbid the extension of the same explanation to those cases in which inflammatory products were inoculated, since in these too local inflammation was excited. They do, therefore, remove the necessity of assuming any specific inoculation to produce the disease, although it has at the same time been found that the injection of fluid from an actually tuberculous organ produces the same infection more readily and certainly. Such cases do not shut out the possibility of some specific morbid poison entering through the air, since the wounds and injuries were always open. In some instances when the setons were steeped in carbolic acid, and in others where subcutaneous fractures of bone were effected, no tuberculous or other disease resulted. Moreover, these animals were in all cases living under very bad hygienic conditions, such as predispose, in man at least, to tubercular disease.

The local changes caused by the irritating substance are regarded by Dr. Sanderson as of great importance, and are thus described by him. There may be, but is not always, suppuration: the primary and characteristic local lesion consists in the development at the seat of injury of granulations or nodules composed of the "adenoid tissue" of His, that is, having what he regards as the anatomical structure of tubercle. Other experimenters have laid much weight upon the caseous degeneration of the products of inflammation, a change which is said to be particularly common in rodent animals; but this certainly does not always occur any more than suppuration. The next step (still following the same authority) in the morbid process is the infection of the lymphatic glands in relation with the seat of primary lesion: they become enlarged, indurated, and caseous. These changes are thought by Dr. Sanderson to be essential steps in the production of general tuberculosis. The infective material he believes to be taken up secondarily by the veins, and having thus entered the systemic circulation, to be distributed by the arteries to all parts of the body. The products of artificial tuberculosis resemble generally those of the spontaneously arising disease in the human subject. Beside granulations in the lungs and on the serous membranes there occur enlargement of the liver and spleen from interstitial growth; and secondary changes, marked by enlargement, induration, and caseation, of the lymphatic glands connected with the diseased organs.

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* Burdon Sanderson: "Tenth Report of the Medical Officer of the Privy Council for the Year 1867." London, 1868. Wilson Fox: "The Artificial Production of Tubercle." London, 1868.

Deductions from the Experimental Results.—These experimental results have modified very materially though indirectly the doctrine of tubercle. They seem to show, in the first place, that secondary tuberculosis may have its starting-point in some simply inflammatory affection; that is to say, there need not be any original "deposit of tubercle" in the sense formerly understood. Secondly. It is not necessary that the inflammatory products, in order to produce secondary tubercle, should pass through any particular kind of degeneration or "caseation." Thirdly. Though it is shown that no specific inoculation is necessary for the development of tuberculosis, it is not proved that some miasma or atmospheric influence is not concerned in its production, or that mal-nutrition does not favour it. The same uncertainty exists here as with regard to the origin of pyæmia.

The theory suggested by these results may be thus summed up. Tuberculosis is generally, if not always, a secondary disease, following some local inflammatory affection; but while several processes, such as induration, suppuration, caseation of inflammatory products often concur, the conditions under which this secondary affection follows are at present unknown; since it is quite certain that all the lesions described as the precursors of general tuberculosis may occur without the development of this disease. The question is equally unsolved whether the infective substance in tubercle (elaborated within the body or introduced from without) is something *specific*, or whether the effects it is supposed to produce are dependent on mere mechanical irritation and obstruction of vessels. In favour of the theory of specific disease must be urged the fact that intestinal tuberculosis has been induced in cattle by feeding them with scrofulous or tubercular products;* and the same disease is said to have been produced in dogs and poultry, which have been allowed to swallow the sputa of phthisical patients. Klebs obtained the same results experimentally by feeding guinea-pigs with tuberculous masses derived both from men and cattle.† In these cases the disease produced was general tuberculosis, but most conspicuously developed in the intestine and mesentery. Moreover, all observers agree that inoculation with true tuberculous material produces the secondary disease more rapidly and directly than any other kind of inoculation. Altogether the question is one of extreme difficulty, and cannot be said to be as yet set at rest.[*]

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* Chauveau: "Gazette Medicale de Lyons," 1868, p. 550; quoted in Virchow and Hirsch, "Jahresbericht für 1869," vol. i. p. 189.

† Virchow's "Archiv.," vol xlix. p. 291.

Tubercle as the Primary Formation.—On the other hand, there are some pathologists who believe that tubercle is always the primary formation in the diseases called tubercular, and that as in the case of cancer or other morbid growths, the associated changes are secondary. They contend that (*a*) it often occurs without any preceding lesion; (*b*) that lesions, *per se*, seem to have little or no power to produce it, without concurrent mal-nutrition and other circumstances; (*c*) that if it were secondary, *i.e.*, dependent on some previous lesion or infection, it ought after a time to get well, *per se*, as pyæmia occasionally does, if the patient live long enough.

Dr. Wilson Fox has lately urged, that in pulmonary phthisis all the changes are dependent upon primary formation of tubercle and accompanying inflammation. Caseation he believes not to take place without the presence of tubercle, and he would therefore restore the old meaning of yellow tubercle.[†]

Schüppel has done the same for the so-called "scrofulous" lymphatic glands, which he believes to be always originally tubercular.[‡]

Finally, it must be said that many authorities believe in a peculiar constitutional state (of the blood or tissues)—*dyscrasia*—which precedes the production of tubercle, and may be regarded as the cause of its peculiar vital properties. This state may be produced by various deteriorating influences, or may be inherited. The fact of heredity again is explained by some as depending upon a peculiar weakness or *vulnerability* of the tissues. These questions it is impossible for us to discuss.

SYPHILITIC PRODUCTS, OR SYPHILOMA.

Constitutional syphilis produces in various organs a species of new growth which is, like tubercle, intermediate between inflammatory products and tumours or new growths, properly so called. It may be in the form of a diffuse infiltration, or of an apparently circumscribed mass. The name *syphiloma* was given to these products by E. Wagner, whose account we chiefly follow. It refers principally to what are called *gummata*.

Syphiloma, when of recent formation, is a soft homogeneous mass of a greyish-pink colour, with either no interstitial fluid, or a very little of mucous character. It varies much in the degree of vascularity. It is only apparently separated by a distinct line from neighbouring tissues. Close examination always shows a gradual transition between the two; the formation of a capsule, if there is one, is always a secondary change. Parts of the organ in which it occurs may be completely replaced by the new growth, so as to produce isolated masses, even when it originates in a diffuse infiltration. The most important elements of a syphiloma are cells resembling leucocytes, and nuclei imbedded in a larger or smaller quantity of fibrous tissue. The cells are roundish, granular, from ·01 to ·03 m.m. in diameter, with usually one nucleus, large in proportion. The nuclei, whether separate or included in cells, have nothing characteristic about them; they preponderate in recently-formed growths and in the external parts of older formations. These elements are imbedded in a fibrous matrix in such a way that usually one cell, sometimes more, is contained in one compartment. In recent growths the matrix does not amount to more than a very delicate stroma, like that of a lymphatic gland, and is only seen when the cells are at least partly removed. In later stages the amount of fibrous stroma becomes larger. Larger alveoli, if present, are often found to be divided in the same way into small compartments. Each visible tumour is made up of a number of smaller nodules.

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[*] For full references to the recent researches see Virchow and Hirsch, "Jahresbericht," 1868, vol. i. p. 189; Ibid. 1869, vol. i. p. 197; and 1870, vol. i. p. 311. A valuable summary is given by Dr. Sanderson, "Edinburgh Medical Journal," Nov. 1869, p. 385. Waldenburg: "Die Tuberculose," Berlin, 1869.

[†] Wilson Fox, discussion at the Pathological Society, March 18, 1873: see "British Medical Journal," March 15, *et seq.*, and "Trans. Path. Soc.," 1873. In the long and interesting debate which followed, the most diverse views were expressed.

[‡] Schüppel: "Untersuchungen über Lymphdrüsen-Tuberculose." Tübingen, 1871. Other references in "London Medical Record," Jan. 22, 1873, p. 33.

The type of structure just described was at first believed by Wagner to be peculiar to syphiloma; but it is plainly not so, since it is essentially that cytogenous or adenoid tissue which is often formed in chronic inflammation. The distinction of syphilitic products from inflammatory is so difficult that some authors (as Billroth) describe them under the latter term. Virchow compares the tissue to that of granulations. The mode of growth and decay of these tumours is more distinctive than their structure.

Involution of Syphiloma.—After a period of growth longer than that of ordinary inflammatory products, the syphiloma begins to waste. The atrophy is accompanied by more or less fatty degeneration. The central parts are first affected and become dry, yellowish, but rather tough than crumbling. In these parts the nuclei and cells are found scanty or absent, an indistinctly fibrillated substance punctated with fat being left. The external parts remain pink and vascular, but in them another change takes place, namely, increase of fibrous tissue. The contrast between the central and external parts goes on increasing, till at length the outside forms a sort of capsule, and the centre softens (though seldom so completely as scrofulous masses) and becomes gradually absorbed. If the absorption is complete only a mass of fibrous tissue may remain, with evidence of loss of substance and contraction. There seems no reason why growth should not go on at the periphery while the central parts are wasting; but probably the growth of which we have evidence on the outside of an old syphiloma, is a sign of the fibrous increase or formation of the capsule. The more active the absorption of the central parts, the more abundant is the capsular connective tissue. In superficial parts atrophy leads to ulceration.

The starting-point of the new growth appears to be always the connective tissue or vessels; never glandular or specialized elements.

Syphilomata are found most often in the skin or subcutaneous connective tissue; then in the muscles, bones, fasciæ, and also in viscera, as liver, kidneys, brain, heart, &c. The existence of actual tumours of this kind in the lungs is somewhat doubtful; though diffuse inflammations doubtless occur in consequence of syphilis.

The distinction from tubercular or scrofulous affections is not always evident; but in general the degenerated or necrotic form of syphiloma is tough, yellowish material, tearing into shreds, but not crumbling. Very seldom (we have seen it once in the liver) is it actually in a pasty condition. On the other hand, the condition may closely resemble the induration of simple chronic inflammation; from which, however, the tendency to degeneration and necrosis distinguishes it.

Lupus.—This form of new growth is not perfectly understood, but seems, like tubercle and syphilitic products, to be intermediate between inflammatory products and true new growths. It is classified by Virchow among tumours, and has a structure not unlike that of granulations. It occurs only in the skin (or rarely mucous surfaces), and consists chiefly of nuclei and cells, with little intervening substance. The nuclei have no special peculiarities, and the cells are mostly like leucocytes, but sometimes larger. According to some authors (Rindfleisch) the sebaceous glands are the original seat of the disease.* It is very difficult anatomically to distinguish the affection from chronic inflammation accompanied by cellular infiltration.

Glanders.—Chronic glanders and farcy, but especially the latter, produce in horses small nodules or "tubercles," compared by Virchow to those of lupus and syphilis. They are less frequently seen when the disease affects man from the acuteness of the attack.

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* Lupus erythematodes is a different disease, probably originating in the glands.

CHAPTER VII.

THE VEGETABLE AND ANIMAL PARASITES OF THE HUMAN BODY.

VEGETABLE PARASITES.

THESE are all cryptogamous plants, and all belong to the order Fungi; but one form, *Sarcina*, was once regarded as an alga. Fungi are cellular or filamentous plants which contain no chlorophyll. Each plant consists of two parts, the mycelium or filamentous layer, which ramifies in the soil or substratum, and the organs of fructification which arise from this. The mycelium has in itself no obvious peculiarities, so that it is difficult or impossible to recognize a species from this alone; the distinctive characters reside in the organs of fructification. These organs produce the spores which are the germs of new individuals. Spores are single cells, usually without a nucleus, and enclosed in a membrane which is remarkable for its power of resistance to decomposition and chemical reagents. Fungi are found in the course of development to pass through very different forms, and their growth is much affected by the circumstances in which they are placed, so that it must remain doubtful whether the forms which we are about to describe are truly distinct species.

I.—FUNGI OCCURRING IN THE BODY, BUT NOT KNOWN TO CAUSE DISEASE.

The mould fungi commonly met with on decaying substances may occur in the human body, but usually on dead or diseased parts, where their presence seems to be the consequence rather than the cause of disease. *Penicillium glaucum* or common blue mould has not been found, unless it be really identical with some of the forms about to be described. The less common form, *aspergillus glaucus*, the blue mould of cheese, occurs in many parts, and has received different names. It grows on diseased portions of skin or on ulcers; sometimes on the nails, and has also been found within the auditory meatus. It may occur on mucous surfaces and in cavities of the lungs. In all cases it forms the ordinary greyish-green covering.

Leptothrix buccalis consists of simple, narrow, very brittle filaments, divided by joints. It is found in every one, in the decomposing masses which collect round the teeth or in the corners of the mouth; on larger masses in the *sordes* of typhus and other diseases. It is always found in the intestine and the fæces, and frequently in the vagina. It is probably without pathological importance, though it has been thought to cause caries of the teeth. It is likely that the vegetative forms or mycelia of several different kinds of fungi are included under this name, and among them that of penicillium glaucum.

Torula cerevisiæ, the fungus of alcoholic fermentation is also found in the contents of every part of the intestinal canal and in diabetic urine. It consists of chains of round or oval colourless cells, which are spores. It has no pathological significance.

Sarcina ventriculi must be placed among the fungi, though it has been classified with the algæ. It consists of round bodies (nuclei or cells) about $\frac{1}{16000}$in. in diameter, which are placed in close apposition, so as to form groups of four, and these groups are again united (though less closely apposed) to form compound groups of 16, 64, and so on. This organism was first discovered by Goodsir in the contents of the stomach, and was at one time regarded as the cause of a special form of disease. It is, however, met with in the stomach in many morbid conditions, as well as in health; less commonly in other parts of the intestinal canal. It has been occasionally found in the urine, in pus, in gangrenous portions of lung, and on one occasion in the brain. It is generally without importance in exciting disease, but seems to have been, in a few cases, the cause of gangrene of the lung. Lately, similar bodies have been found in the blood by Ferrier and Lostorfer.

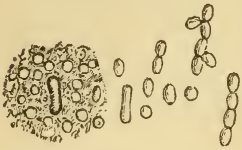
II.—FUNGI WHICH ARE THE CAUSE OF SPECIFIC DISEASES.

These are forms affecting the skin and mucous surfaces. *Trichophyton tonsurans* is the cause of ringworm or tinea tonsurans on the scalp, and the eruption known as herpes circinatus on the body; also of some forms of sycosis. The fungus is most generally seen in the form of spores, which are about $\frac{1}{8000}$ to $\frac{1}{3000}$in. in diameter, very brilliant, and sometimes containing a nucleus. The mycelium is less frequently seen. The spores are found infiltrating the roots of the hairs or collected in masses outside them. They also invade epithelial cells.

Achorion Schoenleinii is the fungus which produces favus or tinea favosa. Its mycelium consists of filaments about $\frac{1}{8000}$in. in diameter, which are often branched, but not articulated or divided by septa. The spores are larger than those of trichophyton tonsurans, being on the average $\frac{1}{3500}$in. in diameter, either free or attached together in rows. The shape of the fully-developed fungus is peculiar, forming an elevated cup (*godet* or favus), the centre of which is pierced by a hair. Spores may be found in epidermic scales as well as in the hair.

Microsporon furfur is the fungus which, affecting the epidermis, produces the disease known as chloasma or pityriasis versicolor. The spores are small, from $\frac{1}{12000}$ to $\frac{1}{6000}$ in., generally nucleated, and with a double contour. There are also filaments, somewhat smaller in diameter than the spores.

Fig. 49.



Drawing of mycoderm of favus. (Partly from Lebert.)

According to many excellent observers the cutaneous vegetable parasites before described are really identical, and are, in fact, merely different stages or forms of the common penicillium glaucum or blue mould; the differences depending upon variations in the conditions of growth. Each form can, it is said, be obtained by cultivation from some of the others. This question is not yet decided, and cannot be in the present state of our knowledge respecting the growth and propagation of fungi.

Oidium albicans is the fungus which is found in the patches known as aphthæ, muguet, or thrush in the mouths of children, and the similar growths which occur on the lips and tongue of adults in certain fevers. It consists of cylindrical, ramified, highly refracting threads, composed of long concatenated cells. Each of the long cells contains several granules. The ends of the threads lose themselves in masses of spores, with a large, sometimes divided, spore cell or sporangium. The spores are round or oval, often concatenated together and form large masses on the epithelial cells.

III.—FUNGI SUPPOSED TO BE THE CAUSE OF EPIDEMIC DISEASES.

The opinion that many diseases which prevail in a particular locality at a particular time (epidemics), or are conveyed from one person to another, with or without absolute contact (infectious diseases), are dependent on a peculiar change in the blood analogous to fermentation (zymosis), is so widely spread that it has led to these diseases being classified in official returns as zymotic. Since the researches of Pasteur and others have shown the intimate connection of ordinary fermentation with the presence and growth, in the fermenting substance, of certain fungi, the further hypothesis has been widely spread, that the zymotic process in the human body is set up by fungi or the spores of fungi, which enter the body from the outside. This supposition unites the "zymotic theory" with the "germ theory" of disease, and were it definitely proved, would afford a satisfactory explanation of most of the phenomena of epidemic and infectious diseases. Without pretend-ing to discuss the question fully, we may enumerate a few of the facts which lend support to some such view. Certain fatal diseases affecting lower organisms, as flies and silkworms, have been shown to be due to infection by means of fungi. The parasitic silkworm disease has been studied with especial care, and is of immense economic importance. Similar parasitic affections have been observed in plants. A still more striking instance is that of the disease of sheep, known as "The Blood,"—a disease closely connected with malignant pustule or *charbon* which affects men. This disease has been shown to be accompanied by the rapid development in the blood of minute organisms, called by their discoverer, Davaine, *bacteridia.* They are very small filaments, which show no spontaneous movement, and disappear with commencing putrefaction, a fact which distinguishes them from the infusoria, which they somewhat resemble. A single drop of blood containing these organisms is sufficient to convey the affection. The same bodies have, moreover, been found in the blood of the human subject affected with malignant pustule.

Though these organisms have been described as fungi or even referred to a particular genus (*Leptothrix*), it seems that they ought more properly to be placed in a group which from a natural history point of view is different, viz., that of the monads or Bacteria; forms possessing only the lowest degree of organization, and standing on the confines of animal and vegetable life. The terminology of these organisms is unfortunately far from fixed, and we have a perplexing variety of terms, such as vibrio, bacterium, micrococcus, microzyme, &c. The common rod-like bodies seen in putrefying animal fluids (bacterium termo) are the most familiar examples. These are the organic forms which always accompany the special decomposition of albuminous substances called *putrefaction;* just as the common Torula accompanies the vinous fermentation of saccharine substances, and may equally be regarded as the cause of the chemical changes. Of late years various forms of these bacteria (as with Cohn we may generically call them) have been found in various morbid products and infective fluids. Thus we have micrococcus vaccinæ, or the bacterium of vaccine lymph (Cohn); micrococcus septicus (Klebs) from septic inflammations, bacillus anthracis (Cohn) from malignant pustule (the bacteridium of Davaine), &c. Others, called "vibriones," have been found by Rindfleisch in pyæmic abscesses.

As an example of these observations, we may refer to the series of researches made by Dr. Burdon Sanderson, or under his direction, on the transference to animals, and from one animal to another of a poison derived from pyæmic inflammations; this poison producing, when injected into the blood or connective tissues of animals, a disease which sometimes resembles pyæmia in the human subject (with metastatic abscesses and a typhoid state); sometimes is like a most intense and rapid blood-poisoning. Still more remarkable is it that the same poison may give rise, under more chronic conditions, to a disease resembling tuberculosis. The poison thus transferred Dr. Sanderson believes to be not chemically dissolved in the infective liquid, but to consist of particles suspended in it; and he further argues that these particles are in all probability organized bodies belonging to the group Bacteria, or, as he calls them, *microzymes*.

In support of this theory, Dr. Burdon Sanderson states that in all infective inflammations (*i.e.*, those produced by infective pyæmic products) in the lower animals, some of the minute organisms, called microzymes, micrococci, bacteria, &c., abound in the exudation liquids, and they are also found in the blood of animals in the state of acute infective fever. These forms he divides into two groups, of which the rod, or *bacterium vibrio*, and the dumb bell, or *bacterium varicosum*, may be taken as types. The latter may also be called "still microzymes," since they have no proper movement like the vibriones. The rods are found in the more intense and acute forms of infective disease; the dumb-bells, as well as spheroidal forms and chains of these, are found in more protracted inflammations. He further believes it will be found that forms such as these exist constantly in the exudation liquids of unhealthy inflammations in man, whether primary or secondary, &c.; and, without stating that they are the *causes* of pyæmic infection, he has no doubt that by their presence in the exudation liquids they modify, in a very decided manner, both the local changes and the constitutional symptoms.* In very rare cases of pyæmic diseases masses of vibriones have been found within the blood-vessels, and even attached to the valves of the heart.† It may then be regarded as established that in several diseases, which have been sometimes described as septic, sometimes infective, organisms, resembling bacteria, are present in the blood and in the inflammatory products. It is moreover admitted by the greatest authority on cryptogamic botany, Cohn,‡ that these *pathogenous* bacteria are distinct from the commoner forms which accompany putrescence (Bacterium termo);—the former being indeed destroyed by the activity of the latter. In what way these minute organisms are connected with the morbid changes which they accompany is yet unknown.

ANIMAL PARASITES.

The lowest forms of animal life known to occur parasitically are certain amœboid creatures, called *Gregarina*, which are found in the intestines of some of the lower animals; but these have not yet been observed in man. Several species of *Infusoria* occur in particular situations on the human body. *Paramœcium coli* is a ciliated organism, occasionally found in the cæcum and colon. Several species of *Cercomonas*, as *C. intestinalis* and *urinarius*, the names of which sufficiently disignate their habitat, are known; and there is an infusorial form, *Trichomonas vaginalis*, the special locality of which is the vagina in cases of purulent discharge.

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* "Trans. Path. Soc.," vol. xxiii. p. 303, 1872.

† Heiberg and Virchow. Virchow's "Archiv.," vol. lvi. p. 407. "Quarterly Journ. Micr. Science," 1873, p. 176.

‡ See "Quarterly Journ. Micr. Science," 1873, p. 156, plate v.

Articulata.—The following *insects* are known as human parasites: —the common flea (*pulex irritans*); the chigoe of the West Indies (*pulex penetrans*); the pediculus capitis; the p. pubis, or phthirius inguinalis; p. vestimenti; the common bug (*cimex lectularius*). Among *Arachnida* we find two species that frequent, the one the epidermis and the other the sebaceous follicles. The *Acarus scabiei* (*sarcoptes hominis*) is a minute whitish creature, about $\frac{1}{100}$—$\frac{1}{50}$in. in size; its head is provided anteriorly with two pairs of mandibular organs, and is furnished with four palpi, or bristles. In the adult state it has eight legs. The female is most commonly met with, being sedentary. She is larger than the male, being $\frac{1}{60}$ or $\frac{1}{80}$in. long. The back is covered with bristles, directed backwards; the two anterior pairs of legs are furnished with discs or suckers; the two posterior pairs terminate in bristles. The male is exceedingly difficult to find, not being fixed to one spot. He is about one-third smaller than the female, and has suckers on the hindmost pair of legs, as well as two anterior pairs. The immature or larval forms have six legs only. The female burrows in the epidermis, and forms minute channels, at the end of which it may often be discovered, and where it lays its eggs. It does not inhabit the vesicles or pustules which constitute the eruption; these are simply excited by the irritative proceedings of the acarus. The *Demodex folliculorum* is proportionally much shorter, about $\frac{1}{125}$—$\frac{1}{75}$ in. long, $\frac{1}{500}$ in. broad; its head has two lateral palpi, with an intervening proboscis, its thorax is supported by four pairs of very short legs, with terminal claws, its long abdomen gradually diminishes to the end, and contains some granular matter and oil vesicles. It inhabits the hair follicles and sebaceous glands in any region of the skin, where it may be found in the healthiest persons. It does not appear to excite any disease.

To the order Arachnida also belong the *Ixodea*, or ticks, which though not always parasitic, attack the domestic animals, and occasionally man; and the harvest bug (*Leptus autumnalis*) which burrows under the skin, apparently for the purpose of laying its eggs (*dictu nefandum*). The irritation produced by these creatures —"*horresco referens*"—is sometimes frightful; the whole surface is covered with red angry-looking lumps, which itch intolerably, so that of scratching there is no end. What is very curious is, that with all this exceeding objective and subjective disorder the cause of offence may remain almost absolutely latent, so that after days of torment a single red speck, running over some part may be the sole indication of the mischief-working agency. If these visible irritants so easily escape notice, how much more may those which generate fevers and influenzas?

The Helminthic parasites are the most important which infest man. Of these the group *Nematoidea* presents us with the following forms:—(1) *Filaria medinensis*, the Guinea worm, from six inches to twelve feet long, about as thick as a piece of packthread. It makes its way into the subcutaneous tissue of the lower limbs and some other parts, where it remains a variable time without exciting any particular symptoms, but when its progeny are approaching the period for their extrusion it makes its way out, or is extracted by hand. The symptoms of this period are sometimes slight, sometimes very severe. If ruptured during extraction the young escape into the cellular texture, and excite an unhealthy suppuration. (2) *Filaria oculi humani*, discovered in the surrounding fluid and in the crystalline lens. (3) *Filaria bronchialis* once found in diseased bronchial glands. Other filaria have been found in the blood and in the urine (spiroptera hominis, dactylius aculeatus). (4) *Trichina spiralis* occurs in the human body, both in the mature and the immature condition. The mature form is found in the intestines of man and other animals. It is a bisexual and viviparous worm. The male is about $\frac{1}{17}$in. long; the female $\frac{1}{12}$in. or $\frac{1}{8}$in. It is tapering at the anterior extremity, blunt at the posterior, and has a perfect alimentary canal divided into pharynx, stomach, and intestine, with distinct sexual organs. The immature or larval form is most commonly met with, and occurs in muscle. It is a minute round worm, $\frac{1}{24}$ to $\frac{1}{36}$in. long, without any obvious internal organs except an intestinal canal with distinct oral and anal openings. It occurs enclosed in a transparent cyst, which is situate in the interior of the fibres of voluntary muscles, separated by the sarcolemma from all surrounding texture. Sometimes there are two or three worms in one cyst. Sometimes the cyst contains calcareous matter, in which case the worm is most commonly dead. The history of this animal is very singular. The immature forms met with in muscle seem to be incapable, in that situation, of complete development. For this purpose it is necessary that the muscle should be eaten by a carnivorous animal, in the stomach or intestine of which the trichina reaches in two days complete sexual maturity; propagation then takes place, and the young embryoes being born alive, immediately commence their migrations through various tissues, till they reach the muscles, where they ultimately settle down. In man the presence of trichinæ in large numbers produces a very formidable febrile disease, which may prove fatal (Trichiniasis); pork containing trichinæ is the usual source of infection. The pathological significance of isolated specimens when encysted is not great; they have often been found in persons who have died of different diseases, and even in those who perished while in health and vigour. (5) *Tricocephalus dispar*, a thin filiform worm, one and a-half to two inches in length. Its anterior two-thirds are quite capillary, and pass rather suddenly into the thicker posterior portion. The sexes are distinct, the male has a long penis invested with a proper sheath. The female produces numerous ova, few of which are probably developed. The worm is found chiefly in the cæcum, adhering by its head to the mucous membrane; it is sometimes solitary, sometimes occurs in great numbers; it does not appear to produce any remarkable symptoms. (6) *Ascaris lumbricoides*, a round worm pointed at both ends, from six to fifteen inches long, of a greyish and sometimes red colour, sufficiently translucent to allow the viscera to be seen, marked by two lateral lines corresponding to the principal vessels, and by two others less distinct, and corresponding to the nervous cords on the dorsal and ventral surface. The head is separated from the body by a slight constriction; at its extremity is the mouth, surrounded by three tubercles. The anus is situated on the under surface, near the extremity of the tail. The reproductive organs consist, in the male, of a single seminal tube of great length, terminating in a reservoir about an inch in length, which communicates with the base of the penis; in the female they comprise the vulva, situated at the junction of the anterior and middle thirds of the body, a vagina five or six lines long, a uterus, which divides into two long tortuous oviducts gradually diminishing to capillary ovarian tubes, which are conspicuous by their whiteness, as they are coiled around the intestinal canal. They inhabit chiefly the small intestines, but roam about occasionally up into the gall ducts, the stomach, the œsophagus, and have even made their way into the nostrils and into the mouth, as happened in a girl under our care. It seems proved by Palm's case[*] that they may perforate the coats of the intestine and get into the peritoneum. Sometimes they are very numerous; one patient passed as many as four hundred and sixty in a fortnight, but this is not common; however, one would rather be inclined to expect from the appearance of one that there were others remaining behind. They have been known to accumulate so as to obstruct the intestines; more commonly they produce only some amount of irritation, or even no symptoms at all. (7) *Oxyuris vermicularis*, a minute, white, threadlike worm, of separate sexes, the male about one and a-half line in length, the female five or six; the former has a spirally coiled tail, the latter a straight tapering and very delicate one. There is a transparent tuberosity on the head, with a kind of alar membrane on each side. They inhabit the rectum in vast numbers, and may crawl out and get, in females, into the orifices of the adjacent canals. They occasion very distressing irritation, perhaps in consequence of their restless disposition, which formerly obtained for them the name of ascarides (*ασκαριζειν*, to leap). (8) *Strongylus gigas*, a formidable large round worm, which attains sometimes three feet in length, and is of a blood-red colour. The male, as usual, is the smaller, is marked by circular striæ and "shallow longitudinal furrows;" at its posterior extremity it has a funnel-shaped pouch, from which a slender penis protrudes. The female has a straighter and more obtuse tail, and at one or two inches' distance from it the vulva. It inhabits the kidneys, and causes more or less destruction of this organ. (9) *Anchylostomum duodenale*, a round worm, a quarter to half an inch long, inhabits in large numbers the duodenum and jejunum, producing hæmorrhage, anæmia, and the so-called "Egyptian chlorosis." It occurs in Italy, Egypt, and the tropics.*

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[*] Würtemb. Corr. Blatt., 1863, 33, 25.

Another Nematoid parasite has been described by Lewis, under the name of *Filaria sanguinis hominis* as occurring in India, in the blood of persons suffering from a peculiar urinary affection called *chyluria*, but sometimes giving rise to no symptoms whatever. It is a small round worm, about $\frac{1}{75}$in. long, and $\frac{1}{3500}$in. in its greatest transverse diameter, of the order Nematoda and family Filaridæ. They are sometimes found in enormous numbers in the blood, and always also in the urine.†

Among the *Trematoda* we are acquainted with four species of distoma, which have been found, though rarely, in the human subject. The *D. hepaticum* and *D. lanceolatum* are both flat lancet-shaped worms, of a yellow-white colour, with two suckers, one of which situated at the head forms the mouth, the other is on the abdomen, and is imperforate. The orifice of the sexual organs lies between these two. They are hermaphrodite. The D. hepaticum is the larger, being eight to fourteen lines in length, and from one and three-quarters to six lines in breadth; its intestinal canal is ramified. The D. lanceolatum, is only two to four lines long and one broad; its intestine is bifurcated. They have been found in the gall bladder and ducts, and in the vena portæ and its branches. *Distoma oculi humani* is of minute size, and has been found in the fluid surrounding a cataractous lens.

To the same order belongs the *Distoma hæmatobium* or *Bilharzia hæmatobia*, a parasite which lives in the blood itself, and feeds on its corpuscular elements. It gives rise to a peculiar form of hæmaturia, especially prevalent in Africa. It was first described by Bilharz in 1851; and has been studied in this country by Dr. J. Harley.‡

CESTOID PARASITES.

General History of Tapeworms.—Before describing the special forms belonging to this order, which includes both the flat intestinal worms or tapeworms, and the cystic-parasites or bladder worms, we must give some account of their history and development,

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* For figure see Weber: "Trans. Path. Soc.," 1867, vol. xviii. p. 274, plate viii. Figs. 1-4.

† T. R. Lewis, M.B., "On a Hæmatozoon inhabiting Human Blood." Calcutta, 1872.

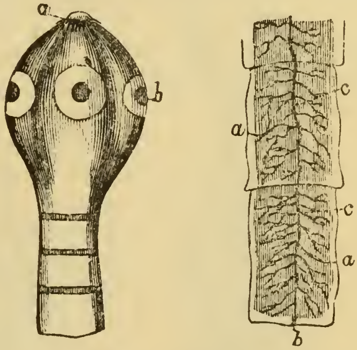
‡ "Med.-Chir. Trans.," xlvii. 55, lii. 379.

without which detailed description would be unintelligible. The flat-jointed worms forming the genus Tænia, properly so called, do not by sexual multiplication produce creatures of their own form. The ova which are developed in each segment of the tapeworms produce, on the other hand, creatures of the cystic type, such as cysticercus or echinococcus, which have no apparatus for sexual reproduction. In or from these forms are produced, however, by a process of gemmation organisms, called scolices, precisely resembling the "head" of the tapeworm; which, when placed in favourable circumstances, develop again into jointed worms. The creature passes through, therefore, two entirely different phases of life—the *strobile* form, as a compound worm capable of true sexual reproduction, and the larval or cystic form as a simple bladder parasite. This process has a distant analogy with the metamorphoses of insects, and a more decided resemblance to the transformations of certain lower animals which are sometimes known as the alternation of generations. But in the case of parasites, the matter is complicated with other conditions. In the first place the two forms of the creature do not live in the same part of the body, the tapeworm always inhabiting the intestines, while the larval form is found in various other organs; and in the second place it does not appear possible for the metamorphosis of the parasite to take place in one and the same animal. The ova or embryoes of the tænia never pass from the intestinal canal to other parts of the same individual, nor do the imperfect scolex-forms pass from the other organs to the intestines of the same animal to undergo further development. It is therefore necessary that there should be two animals (usually of different species), one to act as host to each form of the parasite; and that, generally speaking, one of these animals should serve as food to the other. We shall see how these conditions are fulfilled in the case of the common tapeworm and the echinococcus of the liver. There is, however, one division of this order, that, namely, represented by the broad tapeworm, Bothriocephalus latus (Tænia lata), in which these metamorphoses do not occur, or at least have not been traced. Of those in which they do occur, two, *Tænia solium* and *T. mediocanellata*, are found in the human body, commonly in the strobile form; another, *Tænia echinococcus*, invariably in the larval or cystic form.

Tænia Solium—common tapeworm.—This is a flat-jointed worm, of a whitish-grey colour, attaining sometimes a length of three or four yards. The so-called "head" is at the anterior smallest part of the worm; it is the size of a large pin's head, nearly spherical, with four lateral suckers, between which is a circle of hooks, sometimes double, supported on a moveable organ called the rostellum. The tapering portion of the body next to the head is called the neck; it is imperfectly divided by transverse furrows, but the rest of the worm is composed of distinct segments which increase in length and breadth towards the posterior extremity. The first perfect or mature segment is usually about the 450th. The tapeworm possesses no mouth, digestive or respiratory apparatus, but is furnished with a system of canals opening externally, called the "water-vascular system," the function of which is obscure. Each of the posterior or mature segments, called a proglottis, has complete male or female sexual apparatus, and when impregnated by union with another proglottis, produces within it a large number of eggs, which become developed into embryoes while still within the body of the parent. Each *proglottis* may accordingly be regarded as a single zoological individual, and the whole tapeworm, called technically a *strobilus*, as a compound organism, comparable in some respects to a compound polyp. The *proglottis*, when it has matured its ova, drops off spontaneously from the *strobilus*, and is cast out of the intestine of its host. The embryoes appear to become free by the rupture of the proglottis sac, but are still contained within an eggshell; which is, however, dissolved by the digestive juices of any animal within whose stomach it may by chance find its way. The embryoes of the human tapeworm, dispersed over the ground or in water, are taken up with the food of animals, most commonly of pigs, but sometimes also of cattle. On arriving in the stomach of the pig, the embryo or scolex is set free by the digestion of its eggshell, and aided by an apparatus of six boring-hooks which it bears on its head, makes its way through the walls of the stomach or intestine into some blood-vessel, along which it is carried into the capillaries of some distant part of the body, most commonly (in this species) into the muscles. On becoming stationary, the embryo loses its apparatus of hooks and becomes surrounded by a cyst, while it is itself converted into a bladder lined by a contractible parenchyma. From the inner surface of this parenchyma there buds forth a structure which ultimately becomes a "*scolex*" form and precisely resembles the "head" of the tapeworm; but which remains coiled up within the cyst. In this condition the animal is known as *cysticercus* (*telæ*) *cellulosæ;* and its presence in the flesh of the pig constitutes the disease known as measles. The cycle of transformations is completed, when the cysticercus or measle is transferred by the use of pork as food into the stomach of man or some carnivorous animal, where the cyst being dissolved, the scolex becomes free, and attaching itself to the wall of the small intestine, proceeds to develop into a new strobile or tapeworm form. The fully developed cysticercus varies in size from that of a pea to that of a small kidney bean, presenting an average diameter of ⅓in., though sometimes exceeding it considerably (Cobbold). Its shape is oval in the muscles, round in soft parts or in the cavities of the brain. While the ordinary course of development is that above described, it is possible for the larval or cystic form to become established in the human body, especially in the brain, the intermuscular or subcutaneous connective tissue, and more rarely the liver, kidneys, eyeball, &c.: it is in man usually single, but there may be several individuals, though rarely or never the enormous numbers met with in the pig. The time required for the development of the cysticercus is two months and a-half; the duration of its life is said to be from three to six years. After the death of the animal the body collapses and degenerates more or less completely, while calcareous deposition at the same time takes place, so that there remains at last only a chalky mass surrounded by a fibrous cyst.

The *tænia solium* is a native of Britain, Holland, Germany, Egypt, and the Levant.

Fig. 50.



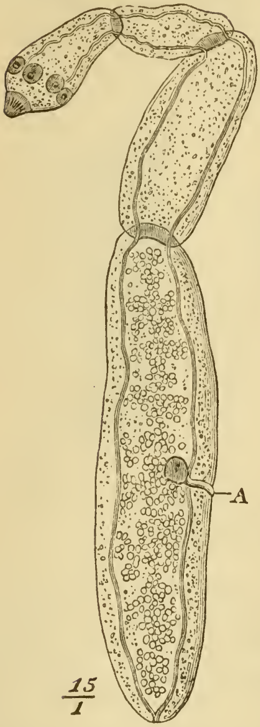
Tænia solium. Head and joints.
(Owen's Lectures.)

Tænia mediocanellata, the hookless tapeworm, considerably exceeds tænia solium in all its dimensions, but especially in the width, even immature segments sometimes measuring half an inch across, and the width does not diminish so rapidly towards the head. The head is without rostellum or hooks, but has four large and distinct sucking discs. The uterus is more complicated than that of the species just described, possessing double the number of lateral branches. The cystic or larval form is somewhat smaller than cysticercus cellulosæ, and of course without hooks. It inhabits the muscles, head, and other organs of cattle, and has never been found in man. This species has been more lately recognized than T. solium, but appears to be in this country equally common. It doubtless finds its way into the human organism through the eating of imperfectly cooked beef or veal. It is regarded by Dr. Cobbold as especially infesting the rich, while T. solium is more common among the poor, who live chiefly upon pork.

Tænia echinococcus, a small tapeworm, seldom exceeding a quarter of an inch in length; composed of three or four segments of which the last exceeds in length all the rest of the organism. The head has a pointed rostellum with thirty or forty hooks and four lateral suckers. The strobile form lives gregariously in the intestines of dogs, but has never been found in man (see Fig. 51.)

The larval form of this tapeworm is much more important, being known in the human body under the names of hydatids, echinococci, acephalocysts, &c. There is no doubt that the early development is like that of the other species of tænia. The six-hooked embryo of T. echinococcus, by whatever means it may find its way into the human intestine, when once there bores its way through the walls and establishes itself most commonly in the liver, though also in other organs; whether it is carried about through the blood-vessels is uncertain. Having become stationary, the embryo passes through a course of development which is at first similar to that of the cysticercus. The hooks drop off, and the caudal vesicle enlarges into a bladder composed of a fibrous external membrane, often laminated (*ectocyst*), and an inner parenchymatous, or blastematous layer (*endocyst*), while there is an adventitious cystic covering, external to all, formed by the condensed fibrous tissue of the organ in which it is lodged. In this state it constitutes what is termed a "hydatid cyst."

FIG. 51.

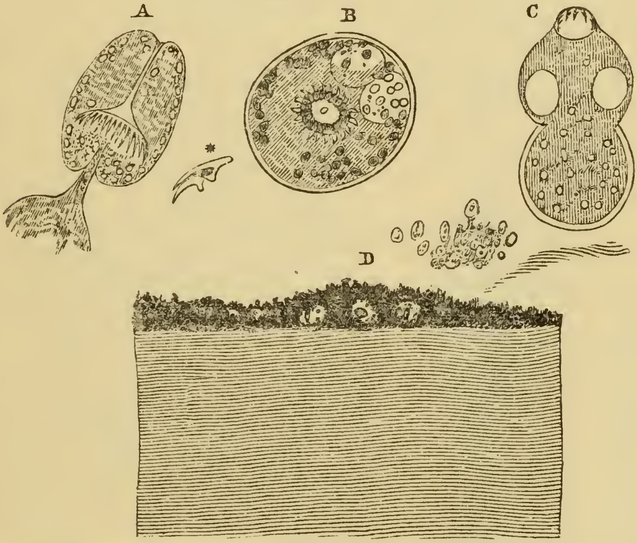


Tænia echinococcus from the dog. A, genital orifice. (After Cobbold.)

Echinococcus Cysts.—When cut into the cyst is found to be filled with a transparent watery fluid, having a specific gravity of 1·008 to 1·013, neutral or slightly alkaline, containing very little, if any albumen (and thus strikingly different from serous effusions), some extractive or fatty matter, and some salts, especially chloride of sodium. Such a cyst may remain single, but may also be multiplied by exogenous or endogenous development; and occasionally a multilocular form is met with, in which there is no large cystic cavity, but a mass made up of a multitude of small cysts not larger than a pea. The endogenous mode of development is the most common in man, and is also seen in the ox. Secondary, or "daughter" cysts, are produced within the primary cyst, and within these tertiary or "grand-daughter" cysts; while even further complications are not unknown. All these derived cysts resemble the parent in having a whitish semi-transparent wall, composed of a laminated *ectocyst* and parenchymatous *endocyst*; but they are of course without the external adventitious covering derived from the surrounding tissues. They also contain the same watery fluid. The structures formed by endogenous multiplication have received the names of Echinococcus hominis, E. altricipariens, E. endogenus, E. hydatidosus, &c. In the exogenous mode of development the original cyst remains simple, though others may be formed on the outside. The forms thus produced are seen commonly in certain of the lower animals, as swine, monkeys, sheep, &c., but very rarely in man; and being thus prevalent when the forms are rare or absent, and being strikingly different in appearance, were formerly described as a distinct species under the names of Echinococcus exogenus, E. veterinorum, E. granulosus, E. scolicipariens; till the identity of the forms denominated by these numerous synonyms was shown by demonstrating the origin of all from tænia echinococcus. The form called echinococcus multilocularis has been observed only in man, and nowhere except in the liver. It may reach a considerable size, forming a tumour as large as a child's head, but contains no considerable cyst, being made up of an enormous number of minute cavities with gelatinous contents, so as greatly to resemble an alveolar colloid cancer, for which indeed the structure has been mistaken. It is particularly liable to central ulceration and softening. (Virchow.)

Production of Scolices.—In the two first-mentioned forms the growth of the echinococcus takes place in substantially the same manner. It is in the substance of the endocyst, whether of the primary or subsequently formed cavities that the *scolices*, which become the "heads" of the adult tæniæ, arise, but by a somewhat indirect process. Small granulations arise in the *endocyst*, which for a certain time grow as if about to form scolices or echinococcus heads directly; but stopping short at a certain stage of development, enlarge and become converted into "brood capsules," or cavities containing a portion of endocystic or parenchymatous membrane; in which a number of scolices are produced. These brood capsules are described as being of the size of a millet seed: they are peculiar to and characteristic of the development of the

FIG. 52.



A, Echinococcus, the head retracted; B, echinococcus, the head and coronet of hooks facing the observer; C, echinococcus, the head extruded; D, section of wall of acephalocyst, with blastematous layer, in which are seen several echinococci.

echinococcus. The origin of the secondary and tertiary cysts is by no means clearly made out. It is thought that they originate by the same process as the brood capsules; these structures becoming enlarged into cysts, while the production of echinococci is, so to speak, deferred; but the subject cannot be discussed here. It is only important to remember that in these secondary cysts, as in the primary, echinococci may or may not be produced from the parenchymatous layer, and that they sometimes remain "sterile," without in any other respect differing from the fertile cysts. The echinococcus heads remain normally attached to the wall on which they are formed, and if they are found free within the cavity, this must be regarded as a sign of death. They measure from $\frac{1}{60}$ to $\frac{1}{100}$in. in length; their body is divided by a constriction into an anterior portion (called the head) which supports the rostellum and suckers; and a posterior portion or base, into which is inserted the pedicle by which the echinococcus is attached to the germinal membrane of the brood capsule. The head bears four suckers and a double crown of hooks, being of course identical with that of the tænia echinococcus. The hooks are important stuctures, as a single one may often be the means of diagnosis. Their size varies somewhat in the two rows; those of the smaller row vary from $\frac{1}{1040}$ to $\frac{1}{1780}$in.; those of the larger row from $\frac{1}{830}$ to $\frac{1}{1780}$in. in length (Cobbold). The head is very frequently seen retracted into the base, so that the animal appears of an oval shape, with an orifice at the part opposite the attachment of the pedicle, and narrow canal leading from thence downward to the middle of the interior, where the circlet of hooks is now seen marking the position of the head. The body of the echinococcus is solid and granular, and contains, especially near the surface, a number of highly refracting calcareous corpuscles, which are very characteristic. The shape and appearance of echinococci show many variations depending upon their death and degeneration, but the hooks and calcareous corpuscles, if seen, are always sufficiently characteristic.

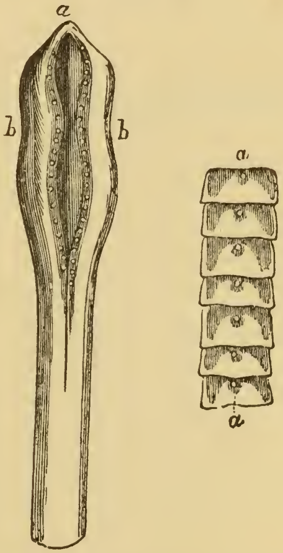
Subsequent Changes of Cysts.—The cysts, both primary and secondary, are liable to degenerate, they become less tensely filled, their walls softer, and more gelatinous, their contents turbid with diffused granulous matter and débris of echinococci. At last they shrivel up into a caseous mass, which is often the seat of calcareous deposit. A variable number of its progeny may decay in this manner, without the parent cyst itself being similarly affected. Inflammation of the external enveloping cyst is the chief cause of the destruction of the primary formation in contact with it; this is sometimes very acute, and leads to the formation of an abscess, which subsequently opens externally, or into some of the adjacent cavities or canals. The débris of the acephalocyst and its contents may be evacuated in this way, and as the cavity contracts and closes a cure is effected. When the inflammation is more chronic, the exudation which is poured out into the cavity of the primary cyst seems to derange the nutrition of the included ones, so that they shrink up and degenerate into mere laminæ of cheesy consistence; these, together with an oily calcareous residue of the fluid contents, which are gradually absorbed, remain in the interior of the primary cyst, which itself wastes and shrinks like its included progeny. In this way another kind of cure of the disease takes place, as the degenerated mass may remain for an indefinite time in the substance of a part without giving rise to any symptoms. Such degenerated acephalocysts have, it would appear, been mistaken for tubercle. The following organs are occasionally the seat of acephalocysts:—the liver κατ’ ἐξοχὴν, the peritoneum, and the underlying areolar tissue, the muscles, the brain, the spleen, the kidneys, the lungs, the bones. They not uncommonly occur in several parts at the same time.

Other species of tapeworm, very rarely found in man, such as *Tœnia nana*, *Tœnia flavopunctata*, &c., need not be further considered here.

Bothriocephalus latus (formerly called Tænia lata), the broad tapeworm, differs from *Tœnia solium* in appearance by the greater proportionate breadth of its segments, and by the structure of its "head," which is without any suckers or hooks, forming a bluntly-pointed, club-shaped mass, $\frac{1}{25}$in. in diameter, slightly flattened horizontally, and furnished with two laterally disposed fossæ or grooves, called *βοθρία* (=*pits*, hence the generic name). The anterior portion of the body is imperfectly segmented, and enlarges in a very gradual manner. The first sexually mature segment or proglottis is about the 600th from the head; the total number of joints is estimated as nearly 4,000.

Each mature proglottis, which may be nearly one inch in breadth, contains both male and female sexual apparatus; the generative orifices being situated in the central line on the ventral aspect, towards the upper part of the segment. The eggs are oval, $\frac{1}{350}$ by $\frac{1}{550}$in., having three shell coverings and a lid-like operculum at one end. They escape from the uterus while the proglottis is still attached, the latter not being discharged whole as in tænia. The embryo has six boring hooks, but is enclosed in a ciliated covering, by means of which it swims freely in the water. It is thought that no intermediate "host" is necessary, and that

Fig. 53.



Bothriocephalus latus; head and joints. *a*, Generative orifices.

(From Owen's Lectures.)

the embryoes are received directly into the human intestine to undergo development; but this point is not quite made out. The *Bothriocephalus latus* is remarkable for its local distribution, being found regularly only in Switzerland and the neighbouring parts of France, parts of Russia, Sweden, and Poland, and certain districts of Germany; less commonly in Holland and Belgium. In this country, as well as out of Europe, it occurs seldom or never, except in persons who have travelled in the countries where it abounds. This peculiarity of distribution is not inconsistent with the conjecture of Cobbold, that some fresh-water fish may be the intermediate host in which it undergoes development; these fish being of all animals least capable of spontaneous migration, and least likely to be transported from one country to another by accidental means.

SPECIAL PATHOLOGICAL ANATOMY.

THE PATHOLOGICAL ANATOMY OF THE NERVOUS SYSTEM.

CHAPTER VIII.

GENERAL OBSERVATIONS.

In no organ or system of the human body is there such an apparent want of accordance and definite relation between the symptoms of disease and the structural derangements and changes produced by it, as in the nervous system at large. This is due to various causes. In the first place, our physiological knowledge of the laws governing the action of the nervous system is not on a par with our acquaintance with the processes operating in other organs; chemistry, physics, mechanics, can be called to aid in the investigation of the healthy and diseased states of the bones, the soft tissues investing them, the thoracic or abdominal viscera; and the structure of these parts is more analogous to what meets the eye of the scientific observer in other departments of nature. It is, then, manifestly impossible, if we are imperfectly acquainted with the laws governing the nervous system in health, that we should be able, sufficiently, to define and appreciate the deviations from the healthy standard; and, in many instances, we must be satisfied to refer for explanation of the symptoms we meet with, to hypothetical analogies, or confess our entire incapability of accounting for the phenomena before us. A correct appreciation of the symptoms of disease is a point which next may be fairly demanded of us if we attempt to explain or seek for morbid phenomena after death; thanks to Sir Charles Bell, Flourens, Marshall Hall, Romberg, and other distinguished inquirers of the present age, much has been cleared up which previously appeared an impenetrable mystery; but our means of physical diagnosis are still but scanty; we are left, in a large number of cases, to form our opinions from the subjective statements of the patient, and where these fail us, as in early childhood, or in certain forms of disease, the greatest empirical experience may be inadequate to offer an explanation of the symptoms, or even to determine their exact relation to cerebral lesions.

In examining the pathology of the nervous system, we shall adopt the succession usually followed by medical writers, and consider, first, the brain and its membranes; next, the spinal cord, with its membranes; third, the cerebro-spinal nerves; the subject will be concluded by a summary of what is known with regard to the sympathetic.

THE DURA MATER.

The intimate relation existing between the dura mater and the cranium renders it peculiarly prone to sympathetic affections propagated from the bone, and from its proximity to the latter it is most likely to be involved in injuries of a traumatic nature. As on its external surface the close contact with the cranium favours a communication of disease from without, so the relation of its internal surface to the arachnoid induces a liability to communication of morbid action from within. A considerable difference exists in the adhesion between the dura mater and the cranium at different periods of life, independently of disease; the connection is very close in childhood, becomes lax in adult life, but is rendered again more intimate and firm in advanced age, as the sutures become ossified, and the bones lose their resiliency. It is especially in the latter period that external injuries are liable to implicate the dura mater as well as the bone; and one of the most common effects of blows or concussions is a forcible separation of the membrane, with hæmorrhage between it and the bone. The clot may, as elsewhere, be partially or entirely absorbed, and we accordingly meet with it in the various stages of metamorphosis. One form of hæmorrhage in this region is connate, and is termed cephalhæmatoma;* it is produced by the pressure exerted during parturition, and is generally found in the form of a tumour, varying in size from a walnut to a child's head, on the parietal bone, presenting during labour. As this, however, is rather an affection of the pericranium we shall revert to it when speaking of the morbid anatomy of the bones. We may state that Nature adopts the same process of limitation and absorption in the case of the external as in the internal hæmorrhage.

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* Deriv. κεφαλὴ, head; and αἱμάτωμα, sanguineous tumour.

INFLAMMATION OF THE DURA MATER, PACHYMENINGITIS.

This may be either acute or chronic; and there are very distinct forms of the acute disease. The first form affects the external layer of the dura mater, or that corresponding to the periosteum, and presents the features of inflammatory action in periosteal coverings generally; it is never of a very active character, and never becomes universal, while in many instances of contiguous inflammation the membrane seems to act as a barrier to intercept its progress. In the first stage the membrane presents a pinky hue, which is irregularly diffused, and evidently has but little tendency to spread. The injection causes the dura mater to assume a more lax and pulpy condition, and it is more readily detached from the adjacent parts. A stage of infiltration and suppuration, or of effusion of lymph, may follow; the latter is the more frequent result of idiopathic inflammation of the dura mater, and may give rise to induration, firm adhesion, thickening, or to new formations, such as the production of bone. In fibrous tissues generally the tendency to lymphatic effusion is rather on the surface, causing an attachment to adjacent parts, whether bone or muscle; in the dura mater, however, interstitial effusion is more frequently met with, on which account the latter is more likely to show the traces of inflammatory action than aponeurotic or fascial expansions. When apparent adhesions occur between the dura mater and the arachnoid, which are not unfrequent, they are rather the effect of inflammatory action and deposition of lymph on the latter than on the former. In such a case the removal of the dura mater cannot be effected without laceration of the arachnoid, or the subjacent grey matter, or connecting bands or shreds are found to be drawn out as the membranes are separated. This form of inflammation is the consequence of some disease, or, more frequently, injury of the cranial bones. Thus it occurs in wounds and fractures of the skull; and from the contiguity of the dura mater to the skull it is very liable to be implicated in external injuries, which may assume the character of the injury to the bone, being cut, punctured, or bruised; or we occasionally meet with laceration of the dura mater from concussion at a distance from the point at which the force was applied. The same causes that give rise to hæmorrhage between the periosteum and bone elsewhere frequently induce extravasation between the dura mater and the inner table of the skull; but they are all of a mechanical nature.

Another frequent cause is inflammation occurring in the internal auditory passages and the cells of the mastoid process. This form is not only secondary, but also of a dyscrasic character; it is met with chiefly in childhood and in scrofulous individuals. The disease may be initiated in the mucous membrane or the bone; and as the morbid process extends towards the cavity of the cranium effusion of lymph or pus takes place under the dura mater, and the brain itself generally becomes involved. The dura mater is often found black and sloughy, especially over the diseased portion of bone; and it is a curious fact, illustrating the propagation of disease from one tissue to another, without actual contact of the morbid process, that there need not necessarily be a perforation of the pars petrosa to induce the inflammation of the dura mater. The same affection, purulent otorrhœa, when of long standing, is liable to induce inflammation in the sinuses of the dura mater; though other injuries, accompanied by purulent discharges, may also give rise to it. This particular form of phlebitis is mainly if not exclusively met with in early life; when the sinuses become inflamed in manhood it is the result of external injury. The dura mater in such a case is found more closely adherent to the skull in the line of the inflamed sinuses and their vicinity than elsewhere, and the contents of the sinuses are fibrinous coagula, while their lining membrane is thickened and deprived of its lustre and smoothness, as in other cases of phlebitis.

The second form of acute inflammation of the dura mater is one affecting its internal or arachnoid surface, and would be described by some authors as inflammation of the arachnoidea parietalis. It is accompanied by the production of very uniform false membranes on this surface, often marked with points of hæmorrhage, which it will sometimes require a little care to distinguish from layers of coagulated blood. This condition is hardly met with except in general diseases, such as pyæmia, fevers, and the like. It is sometimes associated with softened coagula in the venous sinuses, and it may not be easy to say which is the primitive affection.

Chronic Pachymeningitis.—A peculiar form of chronic inflammation of the dura mater has been described by Virchow under the name of Pachymeningitis chronica hæmorrhagica. It is especially a disease of old age, and often accompanies insanity. The first step appears to be the production of an inflammatory false membrane on the inner or arachnoid surface of the dura mater. This false membrane becomes vascularized, and its newly-formed vessels are very liable to rupture, causing extravasation of blood, either between the dura mater and this structure, or else between the layers of the false membrane itself. These processes, set up by the repeated attacks of congestion to which the patients are liable, may frequently recur, and alternate with one another, till masses of considerable thickness are formed; or else a more copious hæmorrhage may result in the formation of a true meningeal apoplectic cyst.*

A gradual absorption of the dura mater from within, and consequent thinning, and perforation, is occasionally observed to follow the growth of tumours from the brain or inner membranes; it may also result from an unusual increase in the size and number of the Pacchionian bodies. Of these we shall have occasion to speak, when considering the relations of the arachnoid.

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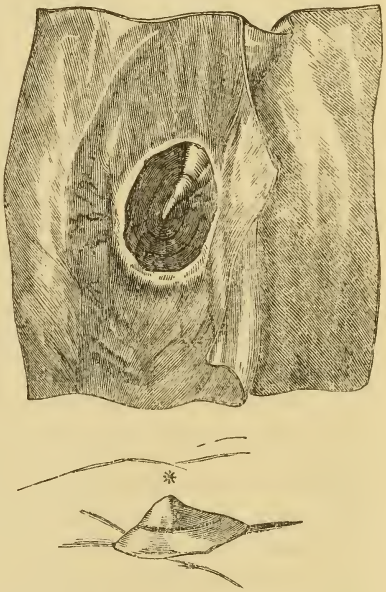
* Virchow: "Die Krankhaften Geschwülste," vol. i. p. 140.

Adventitious Growths of the Dura Mater.—Little is to be said concerning heterologous products occurring in the dura mater; the same cause which accounts for the comparative rarity of idiopathic inflammation in this membrane, suffices to explain why it is less frequently the seat of adventitious growths than the other envelopes of the brain. Fibroid tumours are not unfrequent in connection with the dura mater, from which they are more prone to arise than from other structures within the cranium, according to the prevailing law that the physiological properties of a structure are liable to influence the morbid growths springing from it. In many instances these tumours would come under the head of hypertrophy, rather than of heterologous products.

Both on the internal and external surface of the dura mater we frequently meet with small laminæ of bone, which in many instances may be owing to chronic inflammatory action, of which no other trace is left. Some writers deny that the ossifications found on the inner surface of the dura mater are the products of this membrane; they view them rather as growths belonging to the arachnoid, an opinion to which we demur, both on account of the position in which these bone deposits are found, and from the general endowment of periosteal tissues, to which class the dura mater undoubtedly belongs, to generate bone. These ossifications, which are easily separated from the dura mater, and are generally met with along the falx, must not be confounded with the hypertrophy of the frontal and parietal bones, said to occur during pregnancy, and hence termed, by their discoverer, Rokitansky, puerperal osteophyte. The most remarkable instances of genuine ossification, or of osseous deposit in the dura mater, are occasionally met with in chronic hydrocephalus, where it seems to indicate an effort of Nature to afford extra protection to the

Fig. 54.



A portion of dura mater, exhibiting a mass of bone-like substance of low conical form, attached to the side of the falx cerebri. The patient, twenty-eight years old, had been subject to severe headaches from boyhood. A fortnight before death acute headache supervened, followed by delirium, partial paralysis, and insensibility. There was copious effusion of lymph in the cerebral membranes and ventricles.

* Lateral view of the same. St. Bartholomew's Museum. Series vi. No. 46. Catalogue, vol. i. p. 201.

diseased brain. A delineation of this affection is given in Dr. Bright's medical reports.

Cysts of the kind called dermoid, arising from an involution of the integument at an early stage of development, and containing fat or hair, sometimes occur in the dura mater, and may stretch inwards into the interior of the brain. A remarkable case of this kind is referred to by Mr. Paget, and carefully described by Dr. J. W. Ogle.* It occurred in a child two and a-half years old; the cyst contained a lock of dark hair, with some fatty matter.

Growths of a lipomatous character are occasionally found attached to the inner surface of the dura mater.

We have already seen that the dura mater is frequently subject to secondary inflammation, owing to the extension of scrofulous or tubercular inflammation from adjoining bones; it may thus, as also by extension of tubercular disease from the cerebral tissue, become the seat of tubercular deposit; but the primary deposit of tubercle in the dura mater is probably never seen.

Cancerous Tumours.—Tumours of a cancerous nature not unfrequently grow from the dura mater; and, although the term "fungus of the dura mater" may occasionally have been falsely applied to carcinomatous formations within the cranial bones, still morbid anatomy supplies numerous instances of undoubted cancer of the dura mater. It occurs either in the form of an infiltration of the membrane—in consequence of which the dura mater becomes thickened, and may, by degrees, communicate the carcinomatous infection to the arachnoid, or the bones; or it assumes the form of a rounded tumour, which generally consists of medullary cancer, and pushes its way through the adjoining bone. It is commonly very vascular, and generally occupies a situation near the vertex. When it forces its way through the osseous parietes, these form a ring round it, and the external table of the cranial bone will present a smaller opening than the internal table. Its growth becomes very much more rapid when it has passed the bounds in which it is first kept by the skull, and the soft parts soon become involved and perforated.†

Syphilitic growths of the dura mater are not unfrequently observed. They may grow on the outside, and then cause erosion of the bone; or on the inside, when causing adhesion of the arachnoid they may penetrate through the membranes into the brain itself. They usually form roundish, flattened masses, which may attain a thickness of one-third to half an inch.

Defects and Malformations.—The dura mater rarely exhibits any malformation or defect, beyond those spoken of, except that the falx is occasionally found cribrated, and that in old people the dura mater presents slits in the vicinity of the longitudinal sinus, varying from two to four lines in length, through which the Pacchionian glands have forced their way.

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* "Trans. Path. Soc.," vol. vi. p. 12.

† Cases of cancer of the dura mater may be found in "Trans. Path. Soc.," vol. iv. 1853, pp. 2 and 7.

The records of the Pathological Society of London contain the account of a very rare defect of the falx cerebri, exhibited by Mr. Shaw.* Dr. Bright† also gives a similar case, in which no trace of the process was visible anterior to the tentorium, and it was assumed that the defect, which occurred in a lady thirty years of age, had existed from birth.

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* Reports of the Pathological Society, 1847-48, p. 178.

† Medical Reports, vol. i. p. 150.

CHAPTER IX.

THE ARACHNOID AND THE PIA MATER.

THE traditional doctrine of anatomy, that the arachnoid is a serous membrane of the same character as the pleura or pericardium, has found powerful opponents in Doctors Henle and Kölliker, who have shown that the external lamina is nothing more than an epithelial layer investing the dura mater. It must also be remembered that the arachnoid is entirely dependent for its supply of blood upon the pia mater; and that, consequently, in health as well as disease, the condition of these two structures necessarily bears a very intimate mutual relation. The pathologist has, instinctively as it were, adopted this view, inasmuch as the term meningitis is generally understood to comprise inflammation of the arachnoid and the pia mater, to the exclusion of inflammation of the dura mater, which is not implied in the name; while arachnitis is commonly used to designate disease of the arachnoid, as well as the pia mater, although etymology would not sanction such an interpretation. The best authorities are opposed to over-refinement in these distinctions, and are inclined to deny the limitation of disease to one or the other of the structures under consideration; while on the other hand there are cases of disease affecting the so-called parietal part of the arachnoid or serous lining of the dura mater, without any implication of the true arachnoid or pia mater.

It certainly is more practical, and less likely to mislead the student if we treat of the morbid appearances of the arachnoid and pia mater, under one head, pointing out as we go on those cases and diseases in which the one or the other may appear exclusively or mainly implicated.

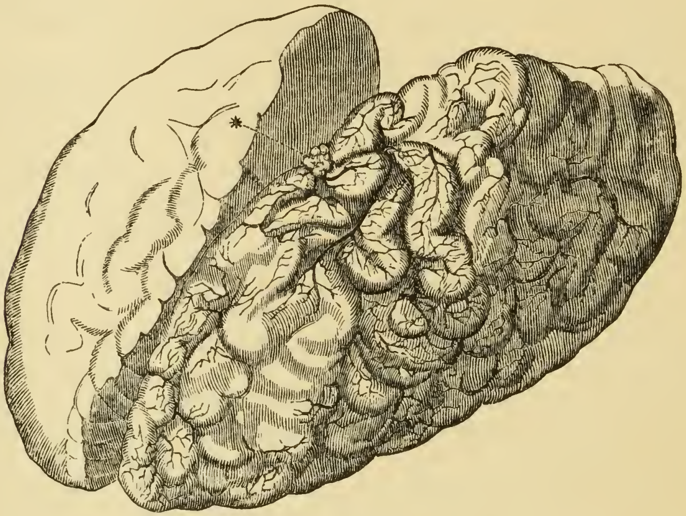
Hyperæmia of the Meninges.—Though the traces of active congestion or hyperæmia of the arachnoid are not visible to the eye after death, we constantly meet with changes in the physical characters of the membrane which are the result of increased action, though the history of the individuals fails to show the occurrence of actual inflammation. Opacity and thickening of the arachnoid, especially on the surface of the hemispheres, are so frequent after the middle period of life as almost to merit being classed under the changes of involution, but they are most marked in habitual drunkards; in delirium tremens they are often the only morbid appearances to be found in the cranial cavity. This opacity is commonly accompanied by more or less serous effusion, which fills the sulci, and raises the membrane from the surface of the brain. Instead of being transparent, and allowing the vessels of the pia mater to shine through, the surface looks milky to a greater or less extent, and more particularly on the surface of the hemispheres. Owing to the obliteration of the sulci by the serum, the affected portion of the cerebrum often looks as if the convolutions were compressed, but on removal of the serum the cause of this appearance at once becomes evident. The subarachnoid effusion may be independent of any affection of the arachnoid, and has been then thought to be due to congestion of the pia mater. It is probable indeed that this is sometimes the case, from the great abundance of vessels in the latter membrane, and their paucity in the arachnoid.

Congestion of the vascular net-work contained in the pia mater is extremely frequent, though we are more often left to infer it, during life, than that we find it after death. It is the very natnre of congestion to disappear in articulo mortis. Still, there is a sufficient number of affections, in which the vessels of the pia mater have been shown to be gorged with blood, without any further accompanying morbid conditions of the adjoining structures; thus we meet with it in cases of pertussis, of fever, of capillary bronchitis, or disease of the heart.

Accumulation of Fluid from Atrophy of the Brain.—From whatever source the serous effusion may be derived, there can be no doubt that among the most frequent determining causes are atrophy and anæmia of the brain. The cranium being a close cavity of virtually invariable capacity, it can be filled only by varying proportions of three substances, viz., blood in the vessels, watery fluid in the subarachnoid spaces and ventricles, and the brain substance itself. Thus Mr. Hilton has shown by experiment that by increasing the blood pressure in the cranial veins, watery fluid is driven out from the cerebro-spinal cavity and the ventricles which are in connection with them; and this is equally true of the fluid in the subarachnoid spaces. Whenever then, from deficient blood pressure through the failure of the heart's action, or from deficiency of brain substance through atrophy, there is, so to speak, a tendency to form a vacuum, watery fluid is poured out to supply the place; though the precise source of this fluid is not yet known. It should be remembered that the subarachnoid or cerebro-spinal fluid is quite different from the serous effusion of other parts of the body. It is a thin watery fluid, of specific gravity 1·007, containing salts but scarcely any albumen; and is thus at once distinguished from "inflammatory effusions" in not being coagulated by heat. The chief conditions in which an excessive quantity of fluid is seen in the subarachnoid spaces are chronic heart and lung diseases; kidney disease; alcoholism; and, as has been said, atrophy of the brain, such as that resulting from old age and starvation. It generally accompanies an excess of fluid in the ventricles and cerebro-spinal space; and a general œdematous condition of the brain. It has been thought that fluid may be sometimes suddenly poured out, causing a kind of acute œdema of the brain, or one form of the so-called "serous apoplexy;" but the reality of this condition is more than questionable. A large quantity of fluid is generally found in cases of sudden death.

There is no doubt that, occasionally, the subarachnoid fluid is attributable to cadaveric changes; it is, therefore, necessary to be circumspect in at once attributing its presence to antecedent morbid action. The amount and position, and, more particularly the concomitant appearances of the pia mater and arachnoid, and the state of nutrition of the brain, must assist us in determining the question in the individual case.

FIG. 55.



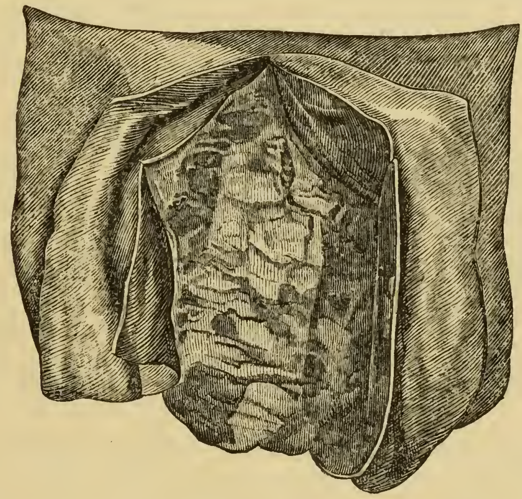
Subarachnoid effusion on the upper surface of the anterior lobe, causing an apparent obliteration of the interstices between the convolutions, and accompanied by increased vascularity.

* Enlarged Pacchionian bodies.

HÆMORRHAGE INTO THE ARACHNOID.

We come now to the consideration of hæmorrhage into the cavity of the arachnoid, a subject which presents a peculiar interest, from being the form of cerebral sanguineous effusion most common in childhood; the reverse condition obtains in adult life, when hæmorrhage within the cerebral tissue is the prevailing form. We have already alluded to one variety of hæmorrhage occurring in the tissues of the head in the new-born infant, which we stated to be the result of the mechanical pressure exerted during parturition; the same cause may give rise to arachnoidal effusion, which then commonly and speedily proves fatal. In this case we find a larger or smaller quantity of fluid blood investing the upper surface of the brain; but, if the child survives the immediate shock of the apoplectic seizure, the effused blood will undergo those series of changes which indicate the tendency of the natural processes to restore the parts to their normal condition. The first step is the formation of a coagulum; the fluid portions are absorbed, then, by degrees, the colouring matter passes through various changes till it also disappears, and we find, in the place of the original coagulum, a membranous formation, which is more or less organized, and may be shown to contain a capillary system of its own. It is a singular fact in the pathology of the disease, to which MM. Rilliet and Barthez were the first to draw attention, that, unlike cerebral hæmorrhage in the adult, it rarely gives rise to paralysis in the child, a circumstance explained by Dr. West,* upon the

Fig. 56.



This drawing represents a portion of the dura mater divided from above, and showing a sac which was filled with coagulated blood. The coats of the sac presented nearly the same thickness as the dura mater.—St. Bartholomew's Museum. Series vi. No. 25.

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* "Lectures on the Diseases of Infancy and Childhood," p. 40. By C. West, M.D. London, 1848.

principle of the blood in the latter instance being almost invariably effused into the cavity of the arachnoid, in consequence of which the effects of the pressure act more uniformly upon all the contents of the cranium. The changes in the effused blood may, however, assume another form than the one just described, and it is important to be aware of the circumstance, as it may simulate hydrocephalus. Instead of the serum being absorbed, it may become enclosed in a false membrane, and remain as a persistent sac, exerting an amount of constant pressure upon the subjacent brain, sufficient to cause flattening of the surface, and to induce, as is generally the case, a considerable impairment of the intellect. When these cysts are once formed they have a great power of passive resistance, and rarely diminish in size.

Superficial or meningeal hæmorrhage occurs more rarely in the adult, except as the result of injury.

Three interesting and instructive cases are given by Dr. Abercrombie,* and we also find a few instances recorded by Dr. Bright.† In the former nothing was found either in the brain or the other viscera to account for the effusion; in one of the cases contained in the Medical Reports there was an hypertrophy of the heart, in another the hæmorrhage was due to the rupture of a small aneurism, in a third no lesions were found, and in two others the hæmorrhage was attributable to a fall, and the viscera do not appear to have presented any disorganization.

Dr. Wilks‡ observes that in all the cases examined by him, the patients had Bright's disease of the kidneys, and our own observation has supplied many instances of the coincidence of the same diseases.

Pacchionian Bodies.—It is not yet decided whether the small nodules, which are found in almost every brain, but become more numerous with advancing age, and have received the name glandulæ Pacchioni,§ are pathological products, or normal constituents. A superficial examination suffices to show that they are not what their first discoverer assumed them to be, conglobate lymphatic glands. They consist of an irregular fibrous network, containing some albuminous granular matter, and generally occupy the vicinity of the mesial line of the surface. They often cause perforations of the dura mater, or of its sinuses, and may thus appear to belong to this membrane, when the brain is taken out in all its envelopes. Their development, at times, is so considerable as even to induce absorption and thinning of the skull-cap. The term arachnoid granulations, applied to them by Louis, is probably as correct a designation as any that has been given them, and we should be inclined to attribute to them no higher importance than that belonging to warty indurations on the surface of the body. Luschka, who has especially investigated the Pacchionian bodies, confirms this view, and describes them as cactus-like projections from the arachnoid, of a fibrous organization, vascular, and covered by a scanty epithelium. He regards them as normal constituents of the membrane, but states that the hypertrophy to which they are liable may be the cause of death by the pressure they occasion. According to Axel Key and Retzius, they are of great importance in regulating the amount of the subarachnoid fluid, being, in fact, the channels by which the excess of this passes into the veins.

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* "Pathol. and Pract. Researches," &c., p. 242.

† Medical Reports, &c., p. 266. See also Mr. Prescott Hewett's paper in the "Medico-Chir. Trans.," vol. xxviii.

‡ "Lectures on Pathological Anatomy," p. 132. London, 1859.

§ "Ant. Pacchioni Dissert. Epistolaris de Glandulis Conglobatis Duræ Meningis." Romæ, 1705.

Other new formations of fibrous tissue are sometimes met with, occurring in a more diffuse form, and producing fibrous masses, which may resemble cartilage in appearance though not in structure. Bony plates are formed in the arachnoid, sometimes as a consequence of inflammation, over the seat of an abscess or focus of softening; sometimes without any obvious cause; in other cases they appear to be ossifications of fibrous thickenings.

Small *epithelial growths* and the fatty masses called steatoma, which are also probably of epithelial origin, are occasionally found on the arachnoid, but have little importance.

Pigment is sometimes found in considerable quantity in the pia mater and arachnoid, with or without melanotic tumours; sometimes associated with pigmentary changes in other parts. Melanotic tumours are distinguished by Virchow* from the groups of melanotic sarcomata and melanotic cancers, and resemble some of those found within the eyeball and on the skin.

MENINGITIS.

Arachnitis is a term used synonymously with meningitis, to designate inflammation of the arachnoid and the pia mater. The nosologist may at the desk draw numerous fine distinctions, and classify symptoms so as to produce a uniform system of morbid processes, but Nature does not bind herself to laws of this description—a remark which applies forcibly to the attempts made to dissever inflammation of the visceral plate of the arachnoid from inflammation of the subjacent pia mater. Whether in the course of pathological research we shall be justified in establishing more minute divisions than we are now able to do is not the question; but we deem it especially our duty throughout this work to place before the student of pathology facts which we may recognize in the dead-house from their having been previously observed by trustworthy inquirers, rather than to show him a maze of systems which have long been the opprobrium of scientific medicine.

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* "Die Krankhaften Geschwülste," vol. ii. p. 119. Figs. 128, 129.

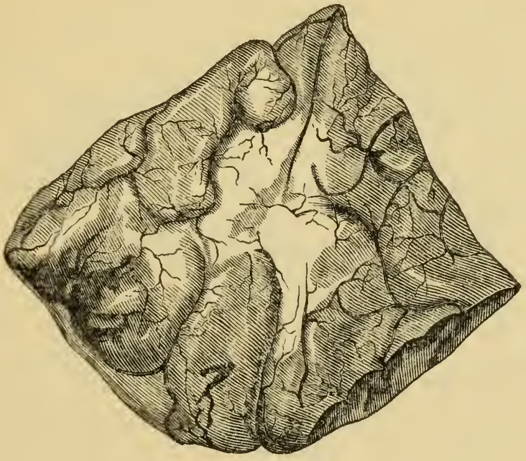
There are then two principal forms of inflammation affecting the arachnoid and pia mater :—Simple meningitis and tubercular meningitis.

SIMPLE ACUTE MENINGITIS.

In this disease three points especially deserve attention—the vascularity of the membranes, the adventitious membrane formed between the arachnoid and the pia mater, and the effusion of serum or pus in the same position. We may, according to the stage and duration of the disease, meet with either of these appearances, or they may be combined in the same individual. If the patient has died in the early stage of the disease we find, on removing the dura mater, that the subjacent membrane shows a great increase of vascularity, which may be so intense as almost to resemble the effusion of blood; the eye and the touch will, however, speedily detect the real nature of the discoloration. The congestion spreads more or less over the surface, or appears in circumscribed patches; on removing a portion of the arachnoid we shall find the congested vessels dipping down with the pia mater between the convolutions. If effusion of lymph has taken place a membranous expansion will be found here and there to intervene between the two meninges, causing a sort of marbled appearance, or bands stretching from one convolution to another. The effused lymph attains the thickness of a wafer and more, and most commonly occupies the upper portion of the hemispheres. The lymph itself dips down into the convolutions, and presents the same variations of density and consistency that this product of inflammation offers elsewhere in proportion to the date of its effusion. Some serous effusion beneath the arachnoid of the base, especially about the optic chiasma, which causes the part to resemble the appearance of jelly, and a small amount of similar fluid (from two drachms to an ounce) in the ventricles, are often found in this form of arachnitis, though by no means necessary accompaniments. The brain, in these cases of simple meningitis, offers no appreciable derangement of structure, though the symptoms during life may have shown very manifestly that its functions were involved. The formation of pus in the course of arachnitis is not an occurrence of frequency, but it is necessary to bear in mind that it is a pathological fact. A remarkable instance of this occurred under our observation at St. Mary's Hospital, in a young woman in whom sudden and unexpected coma supervened, and after thirty-six hours terminated in death. She had previously suffered from otorrhœa, but on her admission gave no evidence of cephalic disease; nor was any direct connection traced after death between the affection of the ear and the meningitis which was found to have caused her death. Here two yellow patches were discovered on each parietal surface of the brain, owing to an accumulation of pus spread out under the meninges; the microscopic examination of the fluid satisfactorily demonstrated the characters of pus.

Cases of meningitis are now and then met with, in which the arachnoid appears perfectly transparent and normal, while there is vascularity of the pia mater, with subarachnoid effusion. We should here be inclined to assume an idiopathic affection of the pia mater, and it cannot be denied that the evidence in favour of primary and independent disease in that membrane is stronger than any arguments adducible in favour of the same disposition in the arachnoid. We cannot blind ourselves to the fact that the latter differs much in its behaviour from the serous membranes of the thorax and abdomen, in the rarity of inflammatory effusion occurring within what we must term the sac of the arachnoid, if we continue to look upon it in the same light as a serous membrane. Exudation on the external surface of the visceral arachnoid is very rarely met with, either alone or accompanying inflammation of the pia mater; and seldom also simultaneously with exudation on the inner surface of the parietal arachnoid, a condition which, rare as it is, is associated only with morbid conditions of the dura mater. These facts show very clearly the pathological independence of the so-called visceral and parietal portions of the arachnoid sac.

FIG. 57.



Portion of upper cerebral hemisphere of a young woman, aged twenty-seven, with purulent effusion under the arachnoid; there were two yellow symmetrical patches, one on each parietal surface, concealing the subjacent convolutions.

Two Forms of Meningitis.—Meningitis sometimes especially affects the vertex, covering the convex portion of the cerebral hemispheres; sometimes, especially the base of the brain, extending over the pons Varolii, the optic chiasma and some adjacent parts of the hemispheres, especially in the fissure of Sylvius, as well as downwards over the medulla oblongata and part of the inferior aspect of the cerebellum. The inflammation also usually finds its way along the velum interpositum and the choroid plexuses into the interior of the brain. We have thus two principal forms of simple meningitis, viz., that of the convex surface of the hemispheres and that of the base. Occasionally both parts are affected at once; but it is very rarely that inflammation is continuous over the whole surface of the brain, the sides and under surface of hemispheres being usually free. When this is the case, or even in meningitis of the base alone, there is sometimes simultaneous inflammation of the meninges of the spinal cord—cerebro-spinal meningitis, a disease which usually occurs as an epidemic, but sometimes in isolated cases arising from special causes, such as excessive drinking, or as a consequence of other diseases.* Meningitis of the hemispheres may be consequent on inflammation of the dura mater, or arise from disease of the brain or some affection of the cerebral vessels. Sometimes it appears to be idiopathic. Meningitis of the base may arise from affections of the bones of the base of the skull, from injury to the head, or sometimes without any obvious cause; but in the immense majority of cases it is dependent on the presence of tubercular growth in the meninges, and will be described as tubercular meningitis. The two diseases differ much in their symptoms, of which we can only say here that meningitis of the hemispheres affects the cortex of the brain, which, as is well known, receives its chief vascular supply from the pia mater, producing violent delirium, or mania; while meningitis of the base is liable to cause paralysis of some of the cranial nerves arising from the base of the brain, and is especially characterized by the symptoms of pressure on the medulla oblongata, pons Varolii, or central ganglia, caused by the large quantity of fluid effused. This liquid effusion affects both the ventricles and the space usually occupied by the cerebro-spinal fluid (subarachnoid space of Hilton), and by dilating the hemispheres, and pressing them upwards against the cranium causes a flattening of the convolutions and obliteration of the sulci, which is very obvious when the skull is opened. When these phenomena occur as the result of rapid disease they constitute the condition known as acute hydrocephalus.

Affections of the meninges often extend, as has been said, into the cerebral ventricles, the morbid conditions of which parts will be considered further on.

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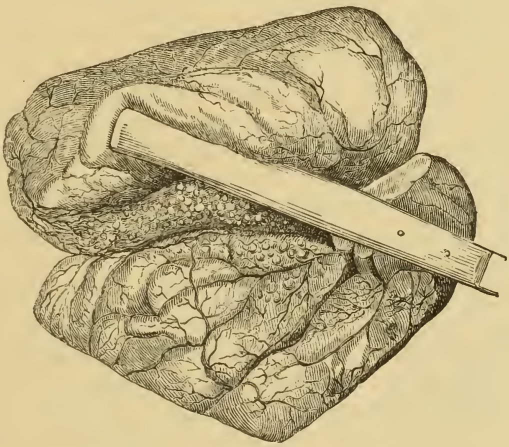
* Payne: "Trans. Path. Soc.," vol. xxi. p. 7. Murchison: "Trans. Path. Soc.," vol. xviii. p. 14. Dickinson: Ibid. xx. p. 22. Sanderson: Ibid. xvi. p. 30.

TUBERCULAR MENINGITIS.

The deposit of tubercle on the pia mater of the brain occurs in the shape of small miliary granules, resembling the Pacchionian bodies in appearance, but differing from them both in their site and their microscopic relations. They are not seen on the free surface of the arachnoid; and, in fact, seem in no way connected with this membrane; a point which establishes a marked difference between it and the serous membranes of the thorax and the abdomen. They are found most frequently deeply within the Sylvian fissure, in the convolutions of the brain, and at the base; they are of the size of pins' heads, and appear in the form of grey granulations, imbedded among a vascular network; they are very rarely found upon the cerebellum. A careful examination is necessary, to prevent their being overlooked; but if not seen on the pia mater itself they may often be found on the minute vessels proceeding from the pia mater into the brain or on the delicate processes of pia mater which dip down between the convolutions. Their presence is always associated with more or less inflammation, as shown by the production of yellow false membranes in the situations pointed out above as the usual seats of meningitis of the base. The amount of inflammation is by no means proportional to the number of tubercular granulations, but seems rather to depend upon the rapidity of the tubercular process; as in very severe cases of acute general tuberculosis there may be numerous tubercles, and very little inflammatory product, while

Fig. 58.

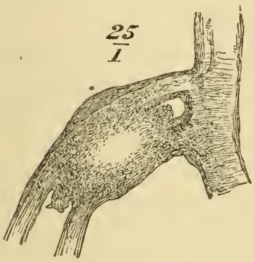


Miliary tubercles in the Sylvian fissure of the brain of a child, aged nineteen months, who died ten days from the first appearance of head symptoms. The white circular spots represent the tubercles, which were surrounded by highly-congested blood-vessels.

in more chronic cases the signs of inflammation are very obvious, but it may require prolonged and careful examination to discover any trace of tubercle, if indeed it be discoverable at all. This affection is well known to be common, and extremely fatal in childhood, and is also the most perfect example of the anatomical features of tubercular growth; so that on both clinical and pathological grounds it is worthy of careful study.

Minute Anatomy of Meningeal Tubercle.—In order to examine the pia mater, in cases of tubercular meningitis, it is best removed by causing a moderately strong stream of water to play between the membrane and the brain. A small portion should then be cut from one of the septa between the convolutions, choosing that part where there is the least possible disease visible to the naked eye. On examination with a low power of the microscope it will at once be seen that the smallest tubercles are in very close connection with small blood-vessels, and rather with the ultimate divisions of arteries than with veins or capillaries. They may be seen to form a distinct projection on the sides of these vessels, very frequently at a bifurcation, and are thus at once distinguished from the fibrous granulations sometimes met with, which are situated in the middle of the vascular meshes. The best specimens are sometimes seen on the small vessels proceeding from the meninges into the brain; which, if the pia mater be removed, as above described, are very perfectly preserved. If the case be one of very rapid disease the tubercular growths are seen quite sharply defined, but otherwise they are often covered by a cloud, which nothing can remove, and which is due to the participation of the contiguous portions of the pia mater proper in the morbid process. Often two adjacent vessels or branches become united, as represented in the figure. When examined with a higher power it can be seen that these projections are really covered by a delicate membrane, which expands from the vascular wall, and that they are in fact situated within a delicate sheath, which also encloses the artery.* This sheath is in fact the peri-vascular sheath discovered by Robin and His round cerebral arteries, and referred by the latter to the lymphatic system. The minute elements of the growth are chiefly small cells (somewhat larger than a red blood corpuscle), with a narrow margin of protoplasma surrounding the nucleus; but there are also many free nuclei and mother-cells containing numerous nuclei. These appearances are taken to indicate the origin of the tuberculous elements from

Fig. 59.



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* See the excellent figure in Virchow, "Die Krankhaften Geschwülste," Bd. ii. 632 (1864–65); also Rindfleisch, "Pathologische Gewebelehre," Figs. 174, 175, pp. 541, 542; Cayley, "Trans. Path. Soc.," vol. xviii. p. 281, Plate ix Figs. 1, 2; Cornil and Ranvier, "Manuel d'Histologie Pathologique," p. 203.

proliferation of the original tissue cells; but there is not yet a perfect agreement as to their precise parentage. Virchow and Rindfleisch described them as due to multiplication of the nuclei normally existing in the outer coat of the artery; while Dr. Bastian, resting on later researches into the anatomical structure of the peri-vascular sheaths, regards them as proceeding from the epithelial cells (endothelia of the Germans) lining these cavities.

Accompanying Inflammatory Changes.—The inflammatory products associated with tubercular meningitis do not usually differ much from those produced by other inflammations. Sometimes, however, they show a much larger quantity than usual of mere granular amorphous matter and fatty molecules; while sometimes they will be found to have undergone a true caseous metamorphosis, and to have become converted into what is called scrofulous or tuberculous matter. The tubercles themselves, it may not be superfluous to repeat, are liable to undergo the same kind of change, and in cases which are not perfectly acute may be found to have become fused into one amorphous mass with the surrounding parts. The rapid progress of the disease never gives time for actual softening or the formation of cavities. Tubercular meningitis, like meningitis of the base proceeding from any other cause, is particularly liable to produce copious effusion into the ventricles, especially the lateral, of the brain, and hence has received the name of acute hydrocephalus; which, in the great majority of cases, means tubercular meningitis.

Causes and Antecedents of Tubercular Meningitis.—This form of meningitis is generally, some pathologists say always, met with in connection with or secondary to tuberculosis or other organs; though we see it occasionally in the idiopathic form, as in the instance from which our delineation was taken, where no tubercular deposit was found in any of the viscera beside the brain. In the former case, the tubercular deposit may take place so insidiously as to offer no marked inflammatory symptoms during life; and it is only on the dissecting-table that the physician becomes aware of the cerebral disease. It must, therefore, be borne in mind, that scrofulous children have a tendency to become afflicted in this manner. Meningeal tubercle being always of the miliary or discrete form is but one variety of that acute tubercular process which has been thought of late years to be always consecutive to degenerative changes of various kinds. Taking the definitions of such changes in a very wide sense, it will be found that meningeal tubercle is only in the rarest cases found to be the original morbid process. The antecedent disease is not always, however, tubercle of other organs, but may be any form of degenerative or scrofulous inflammation, or anything which results in the formation of caseous masses; such as suppurations in lymphatic glands, leading to so-called scrofulous deposits, caseous or scrofulous pneumonia of the lungs, caries of bone, either of the skull or other parts; and perhaps more often than is commonly supposed tubercle of the brain itself. In one case mentioned by Dr. Hillier,* abscesses and pustular eruption of the skin seem to have been the primary affection; and a precisely similar case has fallen under the observation of the editor. Dr. Wilks† states he has in no case, however young, found the lungs free; but the results of other observers are not so absolute.‡ Collections of cases bearing on this subject have been made by Weber,§ Valentin,‖ besides the authors already quoted.

MORBID CONDITIONS OF THE VENTRICLES AND CHOROID PLEXUS.

The inner surface of the cerebral ventricles is so closely connected with the external membranes of the brain as to be seldom unaffected when these are actively diseased. The precise nature of the structure lining the ventricles is not quite clear, some anatomists giving and others withholding the name of a true membrane. The name ependyma may be used without assuming any theory.

Inflammation of the ependyma and choroid plexuses often accompanies meningitis of the base of the brain, whether simple or tubercular. In some cases there is seen merely great fulness of the plexuses and remarkable vascularity of the walls (which are normally very pale) accompanied by a considerable accumulation of fluid (which is distinguished from the normal cerebro-spinal fluid by containing a large amount of albumen and perhaps pus), and some disintegration of the cerebral substance. In more advanced cases there will be inflammatory lymph formed on the surface either of the plexus or the ventricular walls; and the fluid contained in the ventricles will be turbid from the presence of pus and of disorganized cerebral tissue. The ventricles are in all cases greatly distended, and the septum lucidum sometimes broken down. Minute hæmorrhages are extremely common both in the brain substance and in the inflammatory false membranes. This affection is seldom or never found independently of true meningitis of the base of the brain.

Chronic inflammation of the cerebral ventricles seems to occur independently of disease of the cerebral membranes. The ependyma will be found thickened, the subjacent layer of cerebral substance harder than natural. The surface often feels rough to the touch, and looking at it sideways in a good light it may be seen to be covered with minute granulations, which are generally translucent, looking like grains of very white sand or fragments of glass. These granulations may be "as large as hemp seeds, giving the whole the appearance of the leaf of an ice-plant" (Wilks). These granulations are distinctly cellular growths, springing from the ependyma itself. In what seems to be a later stage, the new formations take the character of connective tissue, and distinct fibrous layers may be met with.* Thickening or chronic inflammation of the ependyma is so common in advanced life as hardly to be considered a disease. It usually accompanies some degree of atrophy of the brain, and is associated with accumulation of clear fluid in the cavities. In younger persons it has been thought to be the cause of accumulation of fluid, and has therefore been set down among the causes of hydrocephalus, a condition which is so important as to deserve separate treatment.

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* "Diseases of Children," p. 172. London, 1868.

† "Lectures on Pathological Anatomy," p. 143.

‡ Hillier. *Op. cit.*, p. 161. Other cases recorded in this work are worth notice.

§ "Trans. Path. Soc.," vol. xxi. p. 14

‖ Virchow's "Archiv," vol. xliv. p. 296.

Hæmorrhage into the ventricles is never found except as a consequence of hæmorrhage into the brain itself.

Tumours and new growths are not common, and generally arise from the choroid plexus. Fibrous tumours, fatty tumours, and enchondromata are recorded.

Parasites.—The *cysticercus cellulosæ* has been found lying free in the cavity of the ventricles.† Sir William Jenner has detected *sarcinæ* in the ventricular fluid in a case of cerebral tubercle.

HYDROCEPHALUS.

The etymology of the term indicates the main feature that characterizes this morbid condition. It is truly a dropsy of the brain, and, like dropsies in other parts, is favoured by a relaxed and soft condition of the surrounding tissues. The anatomical peculiarities of the fœtal and infant head at once indicate why it should commence at those periods; while the mal-nutrition upon which it is based, and the further impairment of the important organ in which it occurs, are sufficient grounds for explaining its great fatality, and the infrequency of the children affected with it surviving to reach manhood. Instances are, however, on record, of individuals in whom the parts accommodated themselves to the morbid effusion, and life was prolonged far beyond the period of childhood. The most celebrated case of this kind is that of James Cardinal,‡ who attained the age of twenty-nine, though he had been hydrocephalic from within a fortnight after birth; the circumference of his skull measured at the period of his death thirty-two inches and a quarter.

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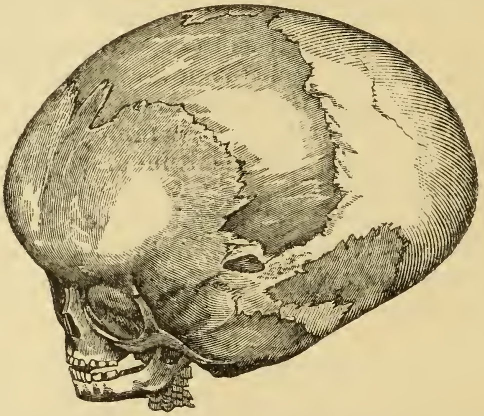
* As the existence and character of the ependyma itself are still matters of dispute, it may not be superfluous to give a few directions for demonstrating its peculiarities. When there is any undue consistency of the parts, no difficulty will be found in stripping off a coherent membrane with some brain substance by inserting the blade or handle of a scalpel under it and pressing outwards. The rough membrane thus obtained should be soaked for a few minutes in water, or for a longer period in weak solution of bichromate of potassium, then laid flat, with the free surface downwards and the cerebral substance removed from the back by firm brushing with a camel's hair pencil. In this way a morbidly thickened ependyma may be always obtained as a distinct coherent membrane, which should be made transparent by immersion in glycerine or otherwise. The method will not succeed, or only with very great care, when the membrane has its normal degree of fineness and fragility.

† Andrew: "Trans. Path. Soc.," xxi. 4.

‡ Dr. Bright's "Medical Reports," p. 431.

The accumulation of serum frequently commences during the last months of fœtal life; it may become a circumstance for the consideration of the accoucheur during parturition, not so much from the increased size of the head presenting an obstacle, because there is even a greater compressibility than usual, but from the head being so soft as not to offer a sufficient fulcrum for the labour pains to act upon; or, in case of operative interference, for the instruments to obtain a sufficient purchase. The child may, however, be born apparently without a blemish, though it does not thrive well, and a few weeks after birth, or at least within the first three years of life, the head appears to increase unduly in size, and the ossification of the fontanelles is retarded. Dr. West states,* that out of fifty cases, symptoms of hydrocephalus were observed in forty-six before they were six months old, and in twelve of these the malady was congenital, and that in nineteen more it came on before the completion of the third month. The accumulation of fluid may amount to as much as ten pounds. The increase in the quantity is greatest during the first months, and diminishes or even remains stationary if the child survives; the circumference of the head corresponds to these relations; thus

Fig. 60.



A hydrocephalic skull from a girl aged eleven years; the enlargement of the skull is effected by its elongation, and by the depression and hollowing of its base. An increase of width appears to have been prevented by the premature and complete closure of the sagittal suture. The coronal suture, and that between the frontal bone and the parietal and the sphenoid, are wide open. The superior walls of the orbits are pressed downwards. The bones generally are thin and light. St. Bartholomew's Museum, sub-series E, 2.

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* "On the Diseases of Infancy," p. 84. 1848.

it is not uncommon to find the circumference to attain as much as twenty-six inches and more, during the first three months, and to vary but little subsequently.

The immediate pathological effect of this secretion is to compress the parts within the skull, and to distend and prevent the due ossification of the cranium; the frontal bone is made to protrude, and the parietal bones, from their yielding character, bulge out considerably on each side, while the intervening soft parts present fluctuation. Ossification takes place, though irregularly, and at a much later period, and we have already seen that a succedaneous deposit of bone is occasionally effected in the dura mater. The sutures of the cranial bones are not formed as in the healthy skull, but are less serrated, and therefore more ready to yield to pressure from within, which they occasionally do after they have closed, in the event of a sudden increase of the fluid. The ossification does not proceed with the normal regularity, but numerous centres form, and thus we meet with more or less ossa triquetra in the line of the sutures. In an almost similar ratio we shall find the brain altered in its relations. If the accumulation has been confined to the ventricular cavities, the entire brain will be distended, and its tissue is generally found softened and pultaceous, from the fluid having infiltrated the cerebral tissues. The distension may be so excessive as to reduce the hemispheres to the thickness of a sheet of paper; but more commonly the parietes give way, and allow the fluids of the two sides to combine; and the pressure may then, as in the case of Cardinal, separate the hemispheres and unfold them, "like the leaves of a book."

It was formerly usual to speak of two forms of hydrocephalus, external and internal, the former commencing in the arachnoid outside the hemispheres, the latter within the ventricles. But there is now no doubt that the disease always originates internally, and that external hydrocephalus is merely a consequence of the ventricular fluid making its way out by rupture of the cerebral substance.

Hydrocephalic Fluid.—The fluid of hydrocephalus is of a limpid and transparent character, or more or less yellowish or opaque; of a specific gravity rather lower than that of the serum of the blood, and containing, therefore, a smaller amount of solid constituents. When there is any opacity, this is found to be due to granular and nucleolar matter, mingled with epithelial *dêbris*. The fluid, when tested by heat and nitric acid, presents the characters of a solution of albumen, and is found to contain chloride of sodium, soda, and traces of salts of lime and potash; and also, according to Dr. Bostock's investigation, urea. The results of his analysis of the hydrocephalic fluid of Cardinal are embodied in the following table:—

Sp. gravity 1011·38

Water	982·6
Albumen	6·0
Chloride of sodium	7·0
Soda	1·4
Urea and osmazome	3·0
Sulphuric acid, lime, potash	a trace.
	1000·0

This composition differs from that of normal cerebro-spinal fluid in containing a larger proportion of solid constituents, viz.—1·8 per cent. instead of about 1·2 per cent., which is normal; in containing more albumen; and in containing urea, which has, however, been found in the cerebro-spinal fluid in several different diseases, especially in disease of the kidneys. The following analysis of normal cerebro-spinal fluid is by Hoppe:—

Water	987·49
Albumen	1·62
Salts and extractive matters	10·52
	999·63

Causes of Hydrocephalus.—It is probable that great accumulation of fluid in the cerebral ventricles may arise from several different causes. In that form which is quite congenital, it would seem *à priori* highly probable that original malformation should be the cause; and such a condition seems to have been discovered by Mr. Hilton, who has most ably demonstrated it in his lectures.* The whole chapter should be read by all who wish to understand the functions and relations of the cerebro-spinal fluid. He there records the fact that in several brains of children dying from hydrocephalus he found the opening which naturally exists below the cerebellum, and between that organ and the medulla oblongata, forming a communication between the fourth ventricle and the spinal canal, completely stopped up; so that fluid contained in the ventricles must be entirely shut in. This condition was found not only in children, but in one case in an adult, though in Mr. Hilton's opinion as a congenital defect. While giving these facts due weight, it must be pointed out that we are yet far from understanding either how the fluid is poured into the cerebral cavities or how it is removed; and that we do not positively know that the spinal canal has any better means of getting rid of an excess of fluid than the cerebral ventricles have. In cases which are not congenital, but where the affection comes on during infancy, there seem to be several influences which must at least be called predisposing causes. Among these are rickets, which, by retarding the ossification of the skull, diminishes its power of resistance; syphilis, scrofulous cachexia, and probably also actual tubercle, though cases arising from the latter cause will generally be classed under the head of acute hydrocephalus. Another cause appears to be the chronic inflammation of the ventricles already described.

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* "Lectures on Rest and Pain," p. 34, Figs. 6 and 7. London, 1863.

We have spoken of hydrocephalus throughout as of a disease of childhood, and it is to early life that it mainly belongs. Several cases are, however, on record of the affection having supervened in the adult when there was no trace of previous cerebral disease. If it be found that the diathesis upon which the disease is engrafted belongs equally to infancy and manhood, we shall easily be able to explain why the more prominent symptom of dropsical accumulation does not present itself frequently in the adult. The firm connection of the cranial bones, and the compression which they consequently exert, offer a mechanical impediment to the effusion which can only take place at the expense of some other contents of the skull. The very congestion of the veins, which for instance exists in cases of uræmia, would militate against it.

Senile Hydrocephalus.—Several instances of an accumulation of serum in the cavity of the arachnoid, or in the ventricles, and not offering any marked traces of active hyperæmia or inflammation occurring in advanced life, may be found in the works of Professor Gölis, Dr. Baillie, and Dr. Watson. They have received the name of senile hydrocephalus, and such are the cases to which the term of serous apoplexy if retained ought to be restricted. The fact of Dean Swift having died of this affection, may serve to fix the subject in the memory. The immediate cause to which this may be most justly attributed is an atrophic condition of the brain, which gives rise to a vacuum, and hence to a discharge from the vessels, of that part of the blood which most readily transudes the coats; the effused serum is peculiarly clear, and its quantity varies according to the extent of cerebral atrophy, from two to six ounces. When we examine into the morbid anatomy of the brain, we shall discuss the origin and causes of atrophy; we have here only to do with one of its products; but it is apparent that in the present instance, as well as in the hydrocephalus of infants, the effusion is a symptom of a deeper-seated malady, and not truly an idiopathic affection. In senile, as well as infantile hydrocephalus, the soft commissures, the septum, fornix, and adjoining parts, are commonly found softened; but it is not always easy to form a positive opinion whether this is a primary or secondary condition.

It may be well also to mention that a sudden effusion of fluid either on the surface or within the ventricles of the brain, the serous apoplexy of some writers, has never been definitely proved to take place, and that the cases in which death is attributed to this cause are for the most part cases of the senile hydrocephalus above described in which sudden death occurs from some other causes, such as disease of the kidneys or heart.

Encephalocele.—Before dismissing this subject we have to advert to a form of congenital hydrocephalus which has received the name of hernia cerebri, or encephalocele, in which, owing to a deficiency in the cranial walls, a portion of the brain and its membranes are protruded. It is analogous to the spina bifida.

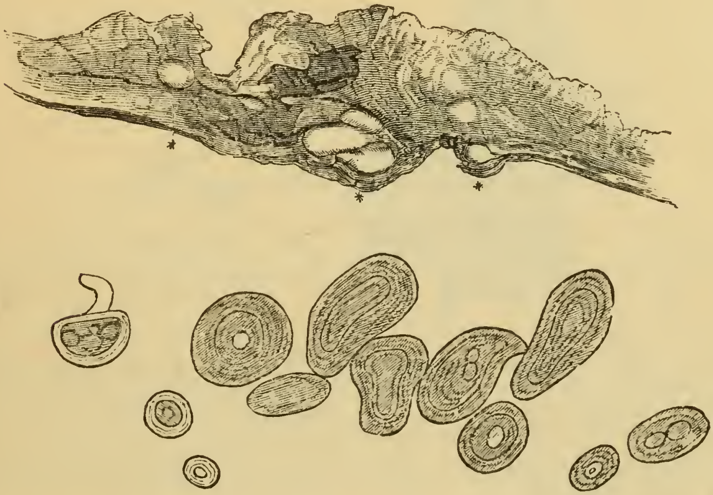
CHOROID PLEXUS.

The venous *rete mirabile* of man, known by the name of the choroid plexus, appears in a measure to possess a vitality independent of the membrane, the pia mater, of which it is an appendix. It is impossible to doubt that it plays a most important part in equalizing and balancing the circulation within the cranium, within the limits of health, and that equally its peculiar relation to the cavity in which it is suspended must give rise to important variations in disease; the physical laws of exosmosis and endosmosis may, without any stretch of hypothesis, be supposed to operate with peculiar vigour, and it is not unreasonable to assume in the choroid plexus a powerful agent of secretion and absorption. The amount of blood in the plexus found after death varies much—at one time it is full and the vessels stand out in relief, at others it is collapsed and contains scarcely enough blood to colour it. There is no necessary relation between the amount of vascularity or exudation, and the congestion or inflammation of the pia mater; thus we may find the surface of the brain covered with a highly vascular pia mater, while the choroid plexuses present an exsanguine appearance; a circumstance which rather confirms our view of their functions.

Concentric Corpuscles.—The greater density of the coats of the vessels in the choroid than in other parts, may account for the rarity of their being the source of hæmorrhage; indurated yellow bodies are, however, occasionally found in them which are referable to former effusion. The morbid appearances most commonly found in the plexuses are round or oval bodies of a yellowish tinge, apparently formed of concentric laminæ, which only become more evident on the addition of acetic acid. They are ordinarily microscopic, but are often found in considerable numbers, and occasionally accumulate into masses of the size of a pea or small nut. Acetic acid acts very slowly upon them, destroying their opacity, but not altering their configuration. They are not found in early infancy, but occur so frequently in advanced life that they almost appear to be a normal constituent. From their behaviour with reagents Virchow calls them corpora amylacea, but the term concentric corpuscles, suggested by Dr. H. Jones, is more appropriate.

Besides these bodies (which are known by giving a brown, dark, or sometimes almost blue colour with iodine) other bodies of more or less concentric structure are met with—the *psammomata* of Virchow. They represent merely an exaggeration of the "brain sand" met with in every choroid plexus. They are especially common on the fringe which extends from the choroid plexuses of the fourth ventricle between the outer surface of the cerebellum and the upper surface of the medulla oblongata, the so-called *flocculus.* These structures have no pathological importance. Mineral acids remove the lime, with effervescence, leaving rings of animal matter.

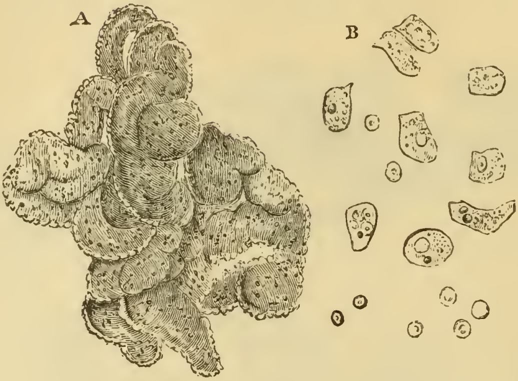
FIG. 61.



The upper part of the figure represents a choroid plexus with several small tumours (at * * *), supposed at first to have been tubercular; they proved to consist of aggregations of concentric corpuscles, cholesterine, and pure oil united by areolar tissue; the concentric corpuscles which are shown below the plexus are magnified 100 diam.

Cysts and Epithelial Changes.—Cysts of the choroid plexus are more frequently mentioned by authors on account of their being visible to the naked eye; they are minute, transparent vesicles, varying in size from a poppy seed to a small pea; and we generally meet with them in brains in which we also find evidence of inflammation of the lining membrane of the ventricles. They sometimes contain a milky fluid, and may present incrustations of a sabulous character. They have been erroneously regarded as hydatids, but there is no evidence of their belonging to this class of parasitic formations; they would rather seem to arise from multiplication of cells, which undergo a colloid degeneration and ultimately liquefy, so as to form a cyst. An hypertrophy of the epithelium, which covers the choroideal vessels, is very commonly observed in advanced age—though we have not been able, as yet, to trace any definite relation between this condition and certain forms of disease.

Fig. 62.



Portion of a choroid plexus, exhibiting a fatty degeneration of the epithelium, from a female aged forty-five, who for fourteen days previous to her death was subject to convulsions and various cerebral symptoms. No morbid condition was observed in the brain, but considerable deposit of oil in the choroid plexuses; in one there was a small lump of fat.

A, The loop of the plexus dotted with oil, 120 diam.; B, the epithelium, containing oil drops, magnified 360 diam.

We also find small fatty tumours on the plexus, as well as a microscopic deposit of fat in the shape of minute oil globules, dotting the surface of the vessels, but for the most part enclosed in the epithelium.

CHAPTER X.

THE BRAIN.—GENERAL OBSERVATIONS.

The question that meets us at the threshold of an inquiry into the pathological conditions of the brain, is whether the amount of blood contained within the cranium in the adult, can vary. There is no difficulty in determining the question in the child, for there, as long as the fontanelles are unclosed by bone, the cerebral circulation necessarily obeys exactly the same laws as rule the general circulation; but when the skull is completely formed, the pressure of the atmosphere is in a measure withdrawn from the contents, and the variation in the amount of blood contained in the cerebral vessels is very much lessened. Still, in applying the law of atmospheric pressure to this subject, we must remember that the column of blood reaching from the heart to the brain is not like the barometer, a single tube, with a vacuum at the closed end, but that the vessels may be compared to a curved tube both ends of which are equally under the control of this law. If it were not so, respiration could not, independent of the heart's beat, exert any influence upon the cerebral circulation. The presence of the ventricles, and of serosity in the cavities and subarachnoid fluid of the brain and spinal canal, is a further indication that there is a provision to meet this species of variation—for it is in obedience to this very law of atmospheric pressure, that we may assume these fluids mutually to assist one another, and as the walls of the cranium cannot collapse, to keep up the balance of the circulation by vicarious action. We have already seen that Nature shows her horror of vacui in senile hydrocephalus, where, if our explanation be correct, the effusion is mainly due to the atrophy of the brain. While we admit, therefore, that the constriction placed upon the vessels of the brain and the peculiar character of the cranial contents, prevent as great a variation in the amount of the fluid contents as takes place in the thoracic or abdominal viscera, we feel assured that a variation does take place, and sufficient to account for many of the phenomena of nutrition and disease. It is important to place this question on a proper basis, as it is one that constantly suggests itself to the pathologist; without a satisfactory explanation, we shall be constantly at a loss to find the proper terms for morbid conditions, and they would themselves appear to contradict our theories. We cannot enter further into this question, but we hope that the suggestions just thrown out may serve to reconcile some conflicting views on the subject; the positive denial of various physiologists, of any variation in the quantity of blood in the brain, apparently supported by such experiments as those of Dr. Kellie, has served not a little to confuse the student. Dr. Burrows[*] investigated this question fully, and from experiments and physiological considerations, arrived at the conclusion, which seems unavoidable to the practitioner of medicine, that the quantity of the blood within the cranium is extremely variable at different times, and under different circumstances. All inquirers are agreed that the relative amount of blood in the different sets of vessels, in the veins, and arteries, varies considerably; and on this point Dr. Kellie's experiments appear to afford conclusive evidence, though the examination of a few bodies would suffice to show the same thing.

There can be no doubt that the most important influence modifying the amount of blood in the brain is the fulness of the ventricles and other cavities of the subarachnoid spaces, and of the lymphatic "spaces" or sheaths surrounding the cerebral blood-vessels, all of which spaces contain a watery fluid identical with the cerebro-spinal fluid, and all communicate with one another. Their fulness, as said above, holds a precisely converse relation to the amount of blood in the brain, a fact which has been established with regard to the perivascular spaces by exact measurements, it having been found by Golgi that they are larger in all conditions of the brain accompanied by anæmia, and smaller in hyperæmic conditions.[†]

Obscure Cerebral Lesions.—We frequently make post-mortem investigations in cases where all the symptoms indicated that death proceeded from cerebral lesion, and where, nevertheless, we are unable to discover any disorganization such as would appear to justify the conclusion that this was the case. A certain amount of hyperæmia in some of the cranial contents may be all that presents itself to us, and even this may be absent. Sir Astley Cooper's experiments[‡] upon rabbits, in which the vertebral arteries were alternately compressed and relaxed, after the carotids had been previously tied, have sufficiently demonstrated the influence of the circulation upon the functions of the brain. The compression invariably produced an instant arrest of respiration, convulsions, and apparent death, and when the finger was removed from the artery the animal gradually recovered. The symptoms closely resembled those of epilepsy in the human subject, to the illustration of which disease they are frequently applied. We have yet to determine the ratio in which mere pressure influences the cerebral functions, as compared with the frequency in which disturbance is excited, and a fatal issue produced by a poisoned condition of the blood, as in uræmia resulting from granular degeneration of the kidneys. It is, however, sufficiently evident that deficient supply of blood to the brain or *anæmia* may be a cause of disordered function, and we are acquainted with two conditions, one temporary the other lasting, by which anæmia of the brain, acute and chronic, may be produced. Spasmodic contraction of the muscles of the small arteries would cause momentary anæmia, and has been assigned as the cause of epilepsy and similar functional derangements; while the thickening of the walls of arteries and diminution of their calibre, in consequence of atheroma, will in the brain as in other parts, cause the supply of blood to be chronically deficient. The former of these conditions will of course leave no signs visible after death, and the latter scarcely any that are unmistakeable.

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[*] "On Disorders of the Cerebral Circulation." London, 1846.

[†] Virchow's "Archiv der Pathologischen Anatomie," vol. li. p. 568. 1870.

[‡] "Guy's Hospital Reports," vol. i. p. 465. 1836.

CONGESTION.

When the congestion of the brain is considerable, the entire organ may present an increase of volume and turgidity. On slicing it the grey matter may exhibit a deeper tinge than usual, but its natural hue prevents the alteration of colour from being very perceptible. The white matter shows an increase of the red dots indicating the blood-vessels, and may, as it does particularly in children, assume a general pinky tint from the same cause. This must not, however, be confounded with the colour imparted to it by the knife as it divides the blood-vessels, and according to the amount of blood contained in them, smears it over the brain surface; by carefully wiping or scraping it we shall be enabled to determine to which cause the colour is due. Scipion Pinel* dwells very forcibly upon the occurrence of congestion of the grey matter of the brain as the main pathological feature accompanying mania. He describes the inner layer of the cortical tissue as presenting a lively red or violet tint, the white matter being less altered in this respect, but also offering a livid hue, with occasional blackish patches, or more or less extended ecchymoses. Congestion of the meninges is more frequently found independently of congestion of the brain than the converse; but we must look for the latter more especially in cases in which death has taken place in consequence of poisoning from opium, in epilepsy and apoplexy, in bronchitis, hooping-cough, in fever accompanied by coma, and in hypertrophy of the heart and granular kidney.

Turgidity of the blood-vessels in the membranes and brain has been observed in the majority of lunatics, independent of other lesions; thus Dr. Webster, in analyzing the records of Bethlehem Hospital, finds this the case in eighty-nine out of one hundred and eight. Local congestions are occasionally met with limited to individual portions of the encephalon.

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* "Traité de Pathologie Cérébrale," p. 193. Paris, 1844.

There can be no doubt that an anæmic condition of the brain, as well as a cachectic state of the blood circulating in it, may induce, in a similar manner, disease and a fatal issue without offering any perceptible lesions. In these cases the fibrous tissue of the brain presents a more deadly white and fewer red spots than in the normal condition, but we possess no means as yet of determining these relations by actual measurement; and in many instances where no organic change has taken place, the tonicity of the arteries may, in articulo mortis, restore the balance of the circulation in such a manner as really to remove all post-mortem effects.

Simple congestion or an early stage of inflammation, appears sometimes to be fatal even when no inflammatory products are formed; and instances of sudden death have been attributed to this cause under the name of apoplexia vascularis. Rokitansky is inclined to think these may be a ratio efficiens mortis, both as the final blow in many acute and chronic diseases, and also quite independently. A remarkable case of fatal cerebral congestion running a very rapid course without any other obvious cause of death is recorded by Dr. Church in the fifth volume of St. Bartholomew's Hospital Reports. (Case viii.)

HÆMORRHAGE.

Congestion is a transition state to numerous cerebral lesions which leave sufficiently perceptible post-mortem effects. The first of these that we shall consider is hæmorrhage, the most frequent cause of cerebral apoplexy, and an affection peculiarly belonging to advanced life; the disposition to it increasing in a direct ratio with the years of the individual. The greatest fatality, according to Dr. Burrows, exists between the ages of sixty and seventy, while it is also found to occur more frequently in males than in females. The following table which has been compiled by Dr. Burrows, clearly exhibits the progressive ratio of apoplexy with advancing age.

Observers.	20 to 30 years	30 to 40.	40 to 50.	50 to 60.	60 to 70.	70 to 80.	Above 80.	Total.
Dr. Abercrombie	3	4	6	7	7	1	0	28
Dr. Bright	4	4	8	4	5	1	0	25
Dr. Andral	3	3	4	6	5	1	0	26
Dr. Rochoux	2	8	7	10	23	12	1	63
Dr. Hope	2	2	9	6	7	11	2	39
Dr. Burrows	2	9	6	8	7	1	1	34
Total in periods of 10 years	16	30	40	41	54	30	4	215

This corresponds in the main with the results obtained from the Registrar-General's reports. To explain the apparent diminution after the age of seventy, we must remember that the total number of living is very much reduced, and that hence the relative number of apoplectic cases is probably even larger than at an earlier age. Cases of apoplexy, even in children, are not unknown, and appear to depend upon disease of the vessels, which sometimes present the appearance represented in Fig. 65.*

The amount of hæmorrhage varies from a spot of the size of a pin's head to an accumulation of many ounces; the former, which may be termed capillary hæmorrhage, is frequently observed in connection with effusions of a more extensive character. but may often be assumed to have occurred during life, where trifling apoplectic symptoms have rapidly passed off under appropriate treatment. It is not generally easy to trace the vessels from which the blood has been effused, and there is also much obscurity as to the actual nature of the morbid action which induces the hæmorrhage. All parts of the encephalon may present apoplectic effusions, but the parts most frequently affected are the anterior lobes, and especially the vicinity of the corpora striata. As a rule the grey structures, including the convolutions of the brain, exhibit the greatest proclivity to the affection, which is in consonance with the known vascularity of these tissues. The structures most removed from the grey matter, as the corpus callosum and the fornix, are least liable to it. Apoplectic effusion is not very frequent in the cerebellum, but it is found to be more rapidly and invariably fatal when it occurs here than elsewhere. Andral's extensive sphere of observation has only presented him with six cases of hæmorrhage into the cerebellum, and in three of these it was associated with hæmorrhage into the cerebrum. It is still more rare, according to Rokitansky, in the pons Varolii and medulla oblongata.*

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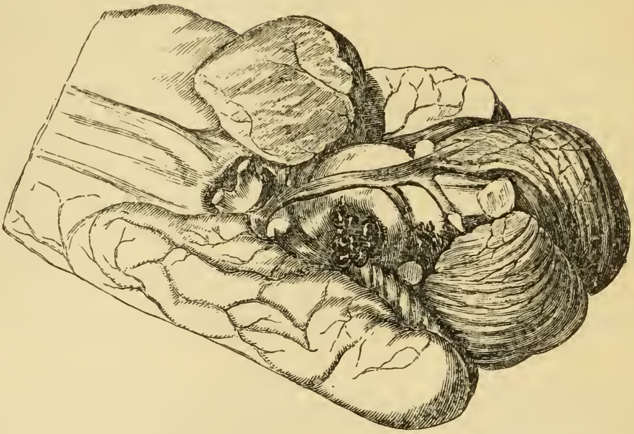
* "Trans. Path Soc.," 1869, vol. xx. pp. 1 and 2, and Fig. 1.

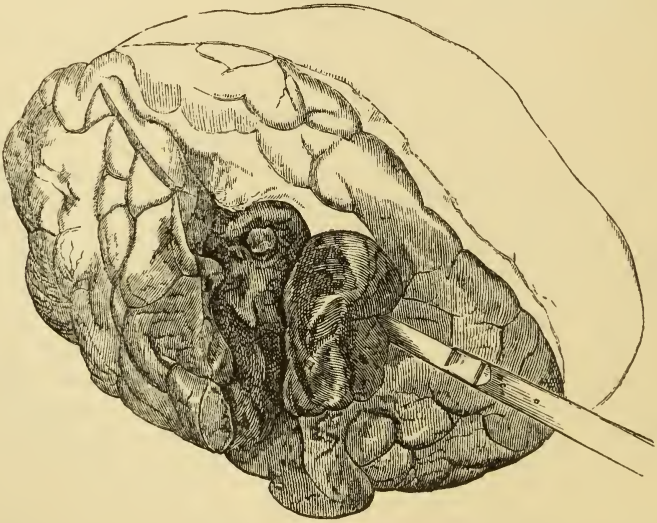
Fig. 63.



Apoplectic effusion upon the left side of the pons Varolii.

Causes of Cerebral Hæmorrhage.—In the causation of apoplectic effusion three elements come into consideration: the condition of

Fig. 64.



Hæmorrhage into the right lateral ventricle and right hemisphere, in a man aged sixty-five. He was brought into St. Mary's Hospital in a state of profound coma, and died two hours after admission. There was a large ragged cavity in the hemisphere, communicating with the ventricle, from which about four ounces of black fluid blood escaped. The corpus striatum and thalamus opticus of right side were much softened. There was no apparent disease of the arteries.

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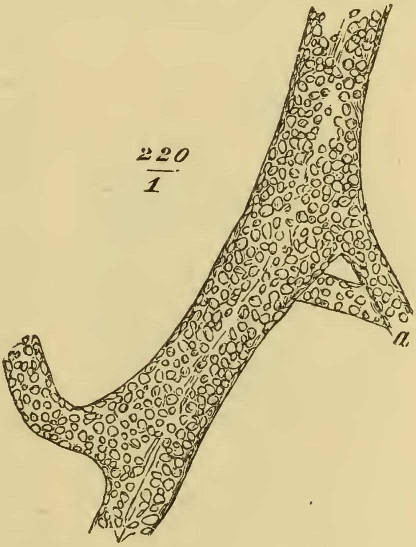
* See cases reported by Church: "Barth. Hosp. Reports," vol. v. Bristowe: "Trans. Path. Soc.," 1857, vol. xviii. p. 18.

the blood, the vascular tension or blood pressure, and the state of the coats of the vessels, and that of the tissues surrounding them. The first, though doubtless important, as necessarily influencing most materially the two latter, we do not as yet possess sufficient data to speak of otherwise than hypothetically. Increase in the tension of blood within the vessels cannot but be a powerful predisposing cause of their rupture ; but that it will not of itself cause hæmorrhage, unless the vessels be diseased, is shown by the fact that apoplectic effusion is not met with in healthy individuals who have died from strangulation, when the blood pressure within the skull must be very greatly increased. On the other hand, apoplexy is most commonly associated with two morbid conditions, which, when occurring together, are distinct evidence of increased arterial tension—viz., granular disease of the kidneys, and hypertrophy of the heart (especially the left ventricle). That there is increased arterial tension in this condition of the body may be shown deductively from the laws of fluid pressure, and is also demonstrated by the sphygmograph ; it is of course obvious that vessels might be strong enough to bear a normal pressure, but give way under one abnormally great.

Fig. 65.



Peri-arteritis. Nuclear proliferation in the outer coat or lymphatic sheath of a cerebral artery from a child.

This is the condition believed to predispose to cerebral hæmorrhage; which, however, had not occurred in this case.

(From an original preparation.)

Condition of the Vessels.—A morbid condition of the coats of the vessels is, however, so much the most powerful factor in the causation of apoplexy, that some pathologists have spoken of it unconditionally as the cause. The principal morbid appearances seen in the vessels in cases of apoplexy are as follows :—(1) Chronic arteritis, giving rise to rigidity and brittleness of the walls, from atheromatous or calcareous deposits; (2) as a consequence of this condition aneurismal dilatations of the smaller arteries, which may be either a kind of dissecting aneurism formed between the middle and the outer coat (lymphatic sheath of some writers), or else true aneurisms composed of all the coats ; (3) simple fatty degeneration of the capillaries and small vessels, which is not always associated with atheromatous or other changes of the larger vessels ; (4) a peculiar nuclear proliferation in the wall of the smaller arteries, sometimes seen in young persons (see Fig. 65).

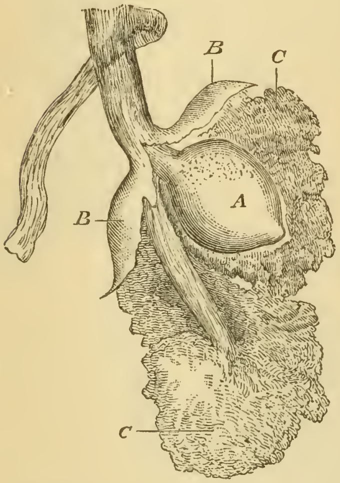
The changes included in the term chronic arteritis will be spoken of under the general head of Diseases of the Arteries, but the occurrence of aneurisms of the cerebral arteries requires some consideration here.

Intracranial Aneurisms.—Aneurisms of the same kind as those occurring in other organs may of course occur in the cerebral arteries, and have been seen, both at various points on the circle of Willis, and on branches within the brain.[*] They are generally associated with disease of the vessel, and hardly ever occur in young persons. When associated with youth and healthy vessels, their formation is not easy to explain, but has been thought to be in some cases due to embolism.[†]

Another kind of aneurism, characteristic of, though not peculiar to, the cerebral vessels, is so important as to deserve special mention.

Miliary Aneurisms of Cerebral Arteries.—MM. Charcot and Bouchard have lately published observations from which they draw the conclusion that the most important and only essential condition of cerebral hæmorrhage is the presence of minute or miliary aneurisms on the cerebral vessels. They attribute the formation of these aneurisms and of more general dilatations which accompany them, not to what is ordinarily called atheroma, or to fatty degeneration, but a peculiar affection of the outer coat of the artery, and of its lymphatic sheath; commencing with a great multiplication of the nuclei which these structures naturally contain, and leading to a fibrous metamorphosis, sometimes accompanied by thickening of these parts. The middle coat loses its muscular fibres, which undergo atrophy, and it is sometimes thickened. The inner coat shows simply multiplication of nuclei. The net result of these changes is, that where there is no marked thickening of the walls, they become weaker, and permit of aneurismal dilatation.

Fig. 66.



Miliary cerebral aneurism and surrounding blood clot.

A, The aneurism; B, remnants of the ruptured perivascular sheath; C, extravasated blood.

(After Charcot and Bouchard.)

The early or nuclear stage of this morbid process, which may be called peri-arteritis, is represented in Fig. 65; it appears to be confined to the intracranial vessels. The aneurisms believed to result from this condition are just visible to the naked eye, varying in size from $\frac{1}{125}$ to $\frac{1}{25}$ inch, and are attached to vessels not more than $\frac{1}{100}$ inch in diameter. They are found in all parts of the brain, but most frequently in the optic thalami and corpora striata, next in the convolutions, and least frequently in the pons Varolii. The number is extremely variable; sometimes only two or three could be discovered, sometimes hundreds. In cases of recent hæmorrhage they look much larger, owing to their being surrounded by a clot which is enclosed within the perivascular sheath; on opening this, the aneurism is found ruptured. The authors observed these aneurisms in every one of eighty-four cases of cerebral hæmorrhage (either recent or occurring some time before death), while no one of the other morbid conditions sometimes associated with apoplexy was observed in more than a certain proportion of the cases.[*]

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[*] Cases may be found in "Trans. Path. Soc.," 1856, vol. vii. pp. 122, 125 (and Fig. 5), 127, 129 (and Plate vi. Fig. 2.)

[†] Dr. Church: "St. Bartholomew's Hospital Reports," 1870.

Recovery from Apoplexy.—The apoplectic effusion is not necessarily fatal in proportion to the amount of blood discharged from the vessels; but the rapidity of the issue appears to bear a relation to the vicinity of the hæmorrhage to the medulla oblongata. The effusion of blood into the ventricles is also marked by being very rapidly fatal. Much probably also depends upon the amount of laceration of the cerebral tissue accompanying the hæmorrhage, inasmuch as the curability of apoplectic effusion seems to be in the ratio of the interstitial character of the discharge. Thus Messrs. Foville and Ollivier have pointed out, both with regard to the encephalon and the spinal cord, that the cure of paralysis resulting from rupture of the nerve-tissue is never complete, but that where the patient is restored to perfect health, the hæmorrhage has mostly separated and compressed the cerebral or spinal fibres. Complete restoration of nervous power is often rendered impossible by the secondary degeneration of the white fibres of the spinal cord which results from continued disease.

Changes in the Effused Blood.—The processes that occur in the blood itself, after it has been effused within the brain, are—the formation of a coagulum, the gradual absorption of the fluid parts of the blood, the formation of an organized membrane around the clot, and the continued absorption of the latter. The rapidity with which these changes occur differs considerably, and depends greatly upon the healthy condition of the surrounding parts. Dr. Macintyre[†] has recorded a case of apoplexy, in which thirteen days after the seizure, the cyst was found fully formed, organized, and nearly empty. On the other hand, Moulin[‡] mentions one of seventeen years' duration, in which a cyst was found containing four ounces of sanguineous fluid. When the effusion occurs in the cavity of the arachnoid, we have seen that cysts also form, but we do not meet with them in the cavity of the ventricles, though there is reason to suppose that the blood may be absorbed from their surface also. According to the degree of absorption, the clot changes its consistency and colour. The clot first assumes a deeper colour, and becomes of a chocolate hue, and from absorption of the serum is rendered hard; the colouring matter is more and more absorbed, a light-coloured fibrinous mass is then seen much contracted from the original dimensions of the clot, and finally this too may disappear, leaving no remains of the hæmorrhagic effusion but the contracted empty cyst, the walls of which are frequently connected by fibrous threads. The cyst itself, in its turn, shrinks up, and finally nothing may remain but a cicatrix. The hæmatoid crystals first discovered by Sir Everard Home, and more recently described by Virchow, are occasionally found in apoplectic clots, together with orange-coloured granular matter. Virchow* states that the earliest period at which he has discovered them, was seventeen days after the injury; their not occurring in recent effusions has been urged as a positive proof regarding the age of a clot.

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[*] Charcot et Bouchard: "Archives de Physiologie," 1868, tome i. pp. 110, 643, 725, planches iii.-v. The same appearances were observed by Heschl ("Wiener. Mediz. Wochenschrift," 1865, Sept. 6 and 9) but not connected with hæmorrhage. A single case was previously observed by Dr. Gull ("Guy's Hospital Reports," 1859, ser. iii. vol. v.) See also Bastian: "Trans. Path. Soc.," 1867, vol. xviii. p. 75.

[†] "Report of Pathol. Society," 1847, p. 11.

[‡] "Traité de l'Apoplexie, ou Hémorrhagie Cérébrale," &c. Paris, 1819.

Changes produced in the Nervous Tissue.—It is manifest that no effusion can take place into the cerebral tissue without a certain amount of disruption of the nerve matter, portions of which may be generally traced within the fresh clot; the greater the previous cerebral softening the more we shall find the brain comminuted. The danger to life, as the recorded cases teach us, is in proportion to the cerebral disorganization, for the tax made upon the powers of the constitution to repair the injury done, is necessarily greater, the more inflammatory reaction is set up; for though the clot possesses in itself a tendency to form a cyst without inflammation, and thus aids in the process of reparation, this cannot suffice to repair the injury done to the cerebral tissue. After the absorption has come to a standstill the cyst or the cicatrix may be borne for years without exciting any new symptoms, and thus, if the individual has suffered from a repetition of apoplectic attacks, we shall find one or more such residuary appearances in a state indicative of the period from which they date. As we occasionally meet with a recent apoplectic effusion that has been effected near the surface of the brain, marked by fluctuation, we find, after the absorption of the fluid, a slight depression in the superincumbent nerve tissue, or a supplementary effusion of serum into the ventricles.

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* See an article on "Blood Crystals," by Dr. Sieveking, in the "British and Foreign Medico-Chirurgical Review," Oct. 1853.

ANÆMIC CONDITIONS OF THE BRAIN.

The converse of the condition which we have just been considering, anæmia, is undoubtedly one of considerable importance, and one that may be assumed to be the cause of numerous morbid conditions; but we have still greater difficulty in determining its existence than that of congestion of the brain. Where it exists the brain presents a generally pallid appearance, and especially the white matter is remarkably deficient in red spots, and more dead-white than normally. It is an important element in the diseases termed white softening and œdema of the brain, which, therefore, especially as in their turn they may give rise to apoplectic effusion, may appropriately be treated of in this place. From what has been said before, it will be at once seen that anæmia of the brain must be accompanied by fulness of the serous and lymphatic channels; that is, by a great abundance of fluid both within the cerebral cavities and generally infiltrated through the brain substance round the vessels; hence the condition will be generally one of *œdema*.

Œdema of the brain is a common condition of the organ met with in ataxic conditions, such as typhus, or puerperal disease, in exanthemata, heart disease, and anasarca. It is common in insanity. Pinel * has described it as the pathological condition characteristic of stupor, or acute dementia. On cutting into a brain thus affected the tissue is found pallid, and the water drips from it, showing a complete œdematous imbibition, The infiltration causes an enlargement of the brain, and consequent flattening of the convolutions, and the pure form of simple œdema is thus distinguished from those in which it is combined with atrophy. The cerebral substance may present various degrees of diminished consistency, till a condition is reached which has been described as white softening.

White Softening.—This is a condition often described, but, it must be confessed, hardly capable of being defined in a perfectly accurate manner, except as an extreme form of œdema. In estimating the degree of this change, it is important to bear in mind the physiological variations in the density of the brain, according to the age of the individual.† It is naturally very soft in infancy, and progressively becomes firmer with the advance of years; and, in old age, as we find a tendency to rigidity of the soft tissues, and to ossification of the cartilages, we discover the brain to present the physiological extreme of density and toughness. The diminished density and consistency or cohesion of a portion of, or the entire brain, constitutes the disease in question. Dr. Bastian states that the specific gravity of softened white matter usually falls to 1·032, but when in its natural condition it is about 1·040.* It is common in children, complicated with hydrocephalus, or as a product of arrested or perverted nutrition, or exanthematic fevers; the affected portions of the brain often being entirely diffluent. In adults it is found in connection with phthisis.

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* "Traité de Pathologie Cérébrale," p. 257.

† See a paper by Dr. Sankey on the "Specific Gravity of the Brain," in the "British and Foreign Medico-Chirurgical Review," Jan. 1853.

White softening is chiefly met with in the parts most remote from the grey matter, as one would expect, if the etiology, as given above, is correct, because they are provided with fewer blood-vessels. Rostan, who was the first to draw attention to cerebral softening, which has since been extensively studied by pathologists, admitted the presence of an inflammatory and a non-inflammatory form; but there is now a general agreement among pathologists to separate this condition from the changes produced by inflammation. Abercrombie and some other writers attribute the form of softening just described under the term of cerebral softening to inflammation; an analysis of the cases given by Abercrombie himself, will, however, be found to support the view of the existence of white softening without the physical signs of inflammation.

INFLAMMATION.

Inflammation of the brain occurs in the various forms, and gives rise to the same products that we find in other organs, modified, of course, in their characters by the anatomical relations of the organ. We find it as an acute and as a chronic disease. Acute inflammation of the brain is not frequently met with in the dead house, in the early stages, nor is idiopathic encephalitis a disease of common occurrence, and many of the cases recorded by older writers resolve themselves into cases of meningitis. It is not our province to inquire into the causes of the peculiarities of diseased action; but we may allude to the circumstance of the brain being so completely withdrawn from physical influences acting immediately upon it, as one of the reasons why idiopathic inflammation should not set up in it, with the same frequency and violence that it exhibits in organs that are more exposed. It is brought on by exposure to the sun's rays in hot summer days or in tropical climates, and may be so rapidly fatal as to produce death before the purulent stage has supervened. Other instances of idiopathic encephalitis are however met with in the early stage, in which no direct exciting cause is traceable. In a case of this kind we find a more or less circumscribed dusky redness in the substance of the brain; the spot generally occupies the upper part of the hemispheres, and, on section, drops of blood may ooze out from the divided surface; there is no necessary change in the consistency of the spot, though it is frequently somewhat softer than the healthy tissue; as the disease advances the exudation of lymph and suppuration ensue, and the colour and consistency of the affected part are modified in proportion. These products are much more frequently the result of chronic and of secondary, than of acute and primary encephalitis. So much so, in fact, that softening, which is the most common effect of inflammatory action, has been treated by many authors as an idiopathic disease.

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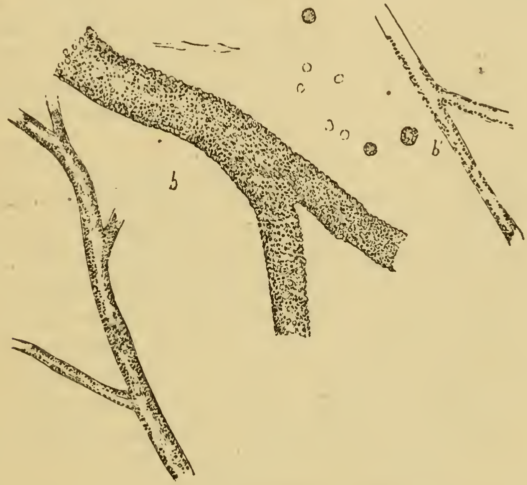
* Bastian: "Specific Gravity of the Brain," in the "Journ. of Mental Science," Jan. 1866, and Reynolds's "System of Medicine," vol. ii. p. 462.

RED SOFTENING.

The condition known as red softening has been a matter of considerable dispute among pathologists, some regarding it as a form of inflammation, others as an infarction due to obstructed circulation. The former view has been till lately that generally accepted.

FIG. 67.



Vessels from the brain of a female aged forty, who died hemiplegic, in consequence of red softening of the right hemisphere.

We find in this condition marked congestion, giving to the affected part a reddish hue; and in the softened tissues the microscope will show, besides broken-down nerve matter, a large number of glomeruli, or granule-cells. The vessels are also often found to be surrounded by collections of fatty granules, either uniformly distributed (Fig. 67), or else in isolated masses. This condition must be distinguished from fatty degeneration of the walls of the vessels. The absence of pus-cells would not be an absolute proof that inflammation had not occurred; for they are not always present [*] in undeniable abscesses, where molecular granules and pyoid bodies may be the sole objects detected by the microscope. We are further borne out in this view by Dr. Bright, [†] who expressly states that we can hardly restrict the term abscess of the brain to those very rare cases in which well-formed pus is found in the substance of the cerebral mass.

We have already alluded to the presence of granule-cells, which are detected by the microscope in degenerative and inflammatory affections of the encephalon. They will here be found in large numbers, and will assist in establishing our post-mortem diagnosis, though, as has been said, not necessarily evidence of inflammation. The necessary disorganization of the cerebral tissue, which must ensue in each of these cases, causes the presence of broken-up nerve matter, which the microscopic specimens will be found to contain. When we have to do with red or apoplectic softening in which hæmorrhage has occurred, we shall also detect blood corpuscles in a more or less altered state. A rough way to determine the presence of softening is, to allow a gentle stream of water to fall upon the suspected part; the softened parts will more or less readily give way and break up. Authors have distinguished from this form of disease another which they call *yellow softening*, but it is now generally agreed that it is but a modification of the condition produced by inflammation; the difference of colour being due to changes in the extravasated colouring matter of the blood.

With regard to the locality of red softening, authors are not agreed as to the preponderance of its occurrence in the grey or the white matter; while Gluge and Durand-Fardel are of opinion that it is more frequently met with in the former; Dr. Bennett's researches lead him to assert that the white matter is the chief seat of this morbid action. French physicians look upon softening of the superficial laminæ of the convolutions as peculiar to dementia, and state that it is characterized by portions of the grey matter adhering to the meninges when they are removed. Laborde has pointed out that this condition often coincides with softening of the corresponding portions of the central ganglia, with which they are connected by nerve-fibres.[‡]

Pathology of Red Softening.—Although granule-cells and collections of fatty molecules round the vessels are very generally seen in cases of red softening, they are not now held to be necessarily evidence of inflammation. They are found in degenerations of the nerve centres, where inflammation is out of the question, and have been observed by MM. Prevost and Cotard in softening of the brain dependent on obstructions of the small arteries and capillaries artificially produced. Granule-cells or glomeruli may represent, there is no doubt, the final stage of fatty transformation of several different kinds of cells. In the brain they have been thought to proceed both from the ganglionic nerve-cells and from the cells of the neuroglia or connective-tissue matrix of the brain. When they occur in the white substance they must no doubt be altered cells of the neuroglia, since this tissue contains no other cells; and even in the grey matter it seems probable that they have the same origin. Since the occurrence of these bodies has, from the time when they were discovered by Gluge, been regarded the main argument for regarding red softening as due to inflammation, many pathologists have of late years discarded the inflammatory hypothesis, and have been led to consider obstruction to the supply of blood as the chief determining cause of this condition. It has now been repeatedly proved by experiment that the obstruction of an arterial branch in any organ produces at first, engorgement of the capillaries in the part supplied by it, and often hæmorrhage. Hence the red colour of parts from which the arterial supply is cut off is not necessarily a sign of inflammation. Similar results follow from obstruction of the veins. Among the causes obstructing circulation are embolism and thrombosis of arteries, embolism of capillaries, and thrombosis of veins and sinuses. Sudden blocking of arteries from embolism produces a rapid and peculiar kind of softening, which will be specially described; but gradual obliteration of the arteries by thrombosis is believed by many authorities to be a frequent cause of red softening. The conditions favouring thrombosis are especially atheromatous changes in the vessels, by which their calibre is diminished and the inner surface roughened; weakness of the heart, which retards the rapidity of the circulation; and, it is supposed, certain states of the blood favouring coagulation. Many observations and experiments supporting these views have been made of late years, especially by French and German pathologists; of which an excellent summary is given by Dr. Bastian in the second volume of Reynolds's "System of Medicine."*

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[*] Lebert: "Physiologie Pathologique," vol. ii. p. 303.

[†] "Reports," p. 171. See also Bennett on "Inflammation of the Nervous Centres," "Edinb. Med. and Surg. Journal," 1842-3.

[‡] Laborde: "Le Ramollissement et la Congestion du Cerveau," pp. 75 and 97. Paris, 1866.

This explanation is in some degree a return to the views of older writers. Thus, Dr. Abercrombie suggested that ramollissement of the cerebral substance was analogous to gangrene occurring in other parts of the body; but, though obliteration of the smaller arteries of the brain may give rise to this change, the name does not seem well chosen, and we are not justified in speaking of gangrene, which means putrefaction, in parts which, though seriously diseased, are by no means withdrawn from the range of vital processes. An important physical symptom of gangrene is remarkably absent in softening of the brain, viz., a fœtid odour.

With regard to the cause of softening, as of cerebral disease generally, it must be mainly sought for in changes directly and primarily affecting the brain; but that peripheral affections may be followed by central disease is forcibly illustrated by the case published by Lallemand* of a soldier, who had been operated upon for aneurism of the right axillary artery. In applying the ligature, the nerve was enclosed, cerebral symptoms followed on the seventh day, and death ensued on the eighth: the post-mortem showed an abscess in the left posterior cerebral lobe. The case is also of interest, as affording proof of the uniformity of the law of crucial conduction.

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* Reynolds and Bastian: Article, "Softening of the Brain," pp. 434-477.

Softening of the brain is looked upon as an essentially fatal disease, though there is no inherent reason why the process of resolution and absorption should not be carried out within the brain as in other organs. It is probable that as our means of diagnosis become more perfect and more refined, our knowledge of this subject will also enlarge; at present we deal rather with the last stages of the disease than with its incipient and more curable features.

ABSCESS OF THE BRAIN.

Suppuration occurs in three forms: we find it occupying the convolutions in the shape of a ragged ulcer, varying in size from a fourpenny-piece, or less, to that of half-a-crown; or the pus is infiltrated through a large extent of cerebral tissue, causing what Dr. Bright terms the diffused abscess; or again, the pus becomes limited, as in other parts of the body, by a membranous expansion, and we then have to deal with the encysted abscess. The consistence and colour of the parts in which exudation and suppuration have been effected, vary according to the extent to which the tissues are involved; the softening may be scarcely sufficient to mark a difference between the healthy and the diseased portions, and it may reach such a degree that the latter are perfectly diffluent; the discoloration, in the same way, will be more or less straw-coloured or reddened, according to the amount of suppuration, the injection of the blood-vessels, and the accompanying exhalation of blood, or its colouring matter.

The greater number of cysts containing pus, that we meet with, are the result of external injury, involving the bone, as if Nature only cared to protect the brain from contact with the external atmosphere. It was already observed by Dr. Baillie, that abscess on the surface of the brain was almost constantly the effect of external violence, but that it was often independent of this cause when formed at a considerable depth within the brain, and that the former was by far the more common form. The cyst itself presents in either case various degrees of thickness and density; the former may amount to half a line, and the latter increase to the consistency of leather.

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* "Recherches Anatomico-pathologiques sur l'Encéphale," &c., vol. i. 123.

The cysts themselves have a laminated fibrous structure, and they are lined with a layer of the pyogenic membrane.

True suppuration of the brain, not arising from injury or diseases of the skull, is mostly of pyæmic origin; the cases in which neither of these modes of origin can be assigned being very few. There are, however, cases on record in which no other evidence of pyæmia has been noticed. Next to direct injury, perhaps, caries of the bones of the skull is the most frequent cause of suppuration in the brain, which may occur with or without meningitis or purulent thrombosis of the cerebral sinuses. Thus it has been observed as a consequence of disease of the internal ear.*

Dr. Gull some years ago examined this subject very carefully, and came to the conclusion that all cases not dependent on injury or disease of the cranial bones, were of pyæmic origin; considering the unexplained cases to be quite exceptional, or due to imperfect observation. In a more recent article in Reynolds's "System of Medicine," an analysis of seventy-six cases is given by himself and Dr. Sutton. Out of this number we find that 40 per cent. depended upon disease of the cranial bones or of the internal ear; in 23 per cent. there was either obvious injury to the skull, or else some history of violence; 13 per cent. were evidently, from the occurrence of suppuration in several parts, cases of pyæmia; in another 13 per cent. there were other diseases, sometimes accompanied by suppuration and softening; while in only 10 per cent. was the origin of suppuration quite unexplained.†

Pyæmic or Metastatic Abscess of the Brain.—Suppuration may occur in the brain, as in other parts, in cases of purulent infection or pyæmia arising from whatever causes. Such abscesses are not, however, very common. They resemble pyæmic abscesses of other parts in the extreme rapidity with which suppuration takes place; a fact which when established by clinical observation, may be an important aid in diagnosis. It is probable that some cases of what appears to be idiopathic abscess of the brain may be due to this cause. For instance, in a case described by Dr. Cholmeley;‡ although no other evidence of pyæmia was established at the autopsy, the patient is said to have suffered from inflammation of the knee-joints, accompanied by swelling and redness, which raises a strong presumption of the existence of pyæmia. But the number of cases in which there has been unquestionable evidence of the existence of pyæmia is not large.

Pyæmic abscesses are said to begin with the formation of a hard, solid, red infarctus, which begins to break down in the centre, and thus forms a focus of suppuration surrounded by highly vascular and hyperæmic tissue. They are very often multiple, and thus in all these respects resemble pyæmic abscesses in the lungs, but differ from them in not specially affecting the superficial parts of the organ, but being rather found, according to Rokitansky, in the deep parts.

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* See cases in "Trans. Path. Soc.," 1867, xviii. 7, by Murchison and Hinton; numerous cases referred to by Gull and Sutton in Reynolds's "System of Medicine," vol. ii. pp. 545 and 572. See also Toynbee: "Diseases of the Ear."

† Gull: "Guy's Hospital Reports," vol. viii. 3rd series. Gull and Sutton: Article, "Abscess of the Brain," in Reynolds's "System of Medicine," vol. ii. p. 544.

‡ "Trans. Path. Soc.," vol xix. 22.

SOFTENING OF THE BRAIN FROM EMBOLISM.—APHASIA.

This seems the proper place to refer to the changes produced in the brain by the sudden cutting off of a portion of its blood-supply. This most commonly occurs, as pointed out by Virchow and Kirkes, through the blocking up of an arterial branch by a small mass of fibrin or a fragment of vegetation detached from the valves of the heart. The fragments thus detached pass up the internal carotid artery, and the branch most frequently blocked is the middle cerebral. Obstruction of the carotid itself usually fails to produce softening, the anastomosis through the circle of Willis being sufficient. The softening will of course vary in extent, but is usually seen on both sides of the fissure of Sylvius, though chiefly in front of it, involving parts of the anterior and middle lobes and the convolutions of the island of Reil, as well as the anterior portion of the corpus striatum. The chief consequence is paralysis of the opposite side of the body, or hemiplegia, and this is often associated with loss of power of speech, a symptom on which we must say a few words.

Aphasia.—Loss of the faculty of language, as distinguished from mere inability to articulate through paralysis, has of late years been shown to have a close connection with disease of the region just indicated, and this only, in the immense majority of cases, when it affects the left, not the right side of the brain. This symptom is thus often associated with right hemiplegia, but may exist without it. Broca has attempted to define still more closely the part which he believes to be the seat of the faculty of language, and locates it in the posterior third of the third frontal (or supramarginal) convolution of the anterior lobe. It appears, however, that a lesion of the corresponding portion of the central ganglia, or of the connecting fibres, or of an adjacent part of the cortex—viz., the convolutions of the island of Reil, may produce aphasia; and even in very rare cases the same symptom follows a corresponding lesion of the right side. Embolic softening is the lesion most frequently discovered, but hæmorrhage or new growth of the same part may produce identical results.

INDURATION.

The converse of ramollissement or induration, appears, like the former, to be an occasional result of a phlogistic process, but like it we must in many instances attribute it to a non-inflammatory change in the nutrient sphere. We find portions of the brain both at the surface and in the deeper seated parts presenting no material alteration beyond an increased density as compared with surrounding parts. This is distinct from the hardened cicatrices resulting from the absorption of apoplectic effusions. A general hardening of the brain accompanied by a livid earthy hue, and an increase of the entire volume of the organ is met with in chronic lead poisoning;* the convolutions are found flattened, the ventricles are compressed, the tissue is dry; and a chemical analysis will detect the presence of sulphate of lead in the brain. Tanquerel des Planches records two cases in which this was done. It is also found in cases of acute lead poisoning.† A similar state of induration and hypertrophy of the white substance is stated by Ferrus and Parchappe and by Dr. Boyd to be found in epilepsy.

Partial or localized induration seems to be the result of a slow inflammatory or hyperplastic process affecting the connective-tissue matrix or neuroglia of the brain. This tissue, and especially the sheaths of the cerebral arteries, undergo a hyperplastic change (as shown by the multiplication of nuclei), and thus compresses and destroys the nervous elements. The process is analogous to that seen in the spinal cord, and will be further considered under that head. It affects the white substance of the brain; viz., either the central portion of the cerebral hemispheres, the corpus callosum, fornix, fimbria, septum pellucidum, tractus opticus, corpora geniculata, crura cerebri, pons, or medulla oblongata, and eventually causes contraction and deformity of the part.

HYPERTROPHY OF THE BRAIN.

Although in adult life when the skull is completely formed, disease cannot cause any alteration in the size of the brain, as it does in other organs, very great variations due to original development are met with; and in early life disease may cause a remarkable increase in the size and weight of the organ; though as this appears to be due to the excessive formation of non-nervous tissue, it must not be spoken of as hypertrophy without a certain reservation.

Specific Hypertrophy of the Brain.—In a case of hypertrophy, on removing the skull-cap, the brain seems to expand, as if it had been previously confined in too narrow a space; the membranes are thin, the convolutions are flattened by being compressed against the bone, and the ventricles are found to contain very little or no fluid. On making a horizontal section, the grey matter is not seen altered, and the naked-eye view displays an increase in the amount of white matter; this, according to Rokitansky, is owing, not to an augmentation in the number of nerve tubes, or their dimensions, but in the excessive development of the intervening and nucleated substance or neuroglia.

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* Tanquerel des Planches: "Traité des Maladies de Plomb," vol. ii. p. 298. Paris, 1839.
† Alfred Taylor on Poisons, &c., p. 133.

Hypertrophy appears to be due to some constitutional cachexia, and is met with chiefly in early childhood. As long as the fontanelles are not closed, it does not in itself involve danger, and even the intellect continues unimpaired; but as soon as the fontanelles have closed, the undue pressure gives rise to numerous cerebral symptoms, none of which, however, are characteristic of this disease. It belongs essentially to the family of rhachitic affections; the frequent coincident distortion of the bones, the swelling of the lymphatic glands, and the general torpor of the system, suffice to establish its relationship. The bloodlessness and dryness of the tissues must assist us in deciding in a doubtful case, whether we have to deal with hypertrophy of this character, or with hyperæmia, hydrocephalus, or œdema, conditions which also may cause the brain to appear too large for its case, and produce a flattening of the convolutions. Nor would it be just to consider as diseased a brain, which, though larger than the average at the age of the individual, exhibits no morbid relations such as those described.

We must also be careful not to confound cases of tumefaction of the brain from softening with hypertrophy; thus we find in Andral's "Clinique Médicale" the account of a post-mortem, in which, owing to this cause, the left hemisphere was so much swollen as to push over the mesial line to the right side. A similar specimen is preserved in the museum of St. Thomas's Hospital.

Individual parts of the cerebrum are very rarely found hypertrophied by themselves. Dr. Mauthner * records a case of hypertrophy of the thalamus opticus of the right hemisphere in a child of three years of age, which, till within three weeks of its death, had enjoyed sound health. She then fell from her chair, striking the occiput, and became paralyzed on the left side. Shortly before death scarlet fever supervened, she became delirious, and died comatose. The thalamus is described as enlarged to the size of a hen's egg, of a lardaceous, dead white appearance on section, without softening of the adjacent parts or effusion; except that the testes were very vascular and the left optic nerve enlarged, no abnormity was discovered. Dr. Mauthner is of opinion that the organic malady had remained latent, until the occurrence of the fall. The same author gives an interesting table showing the weights of the brains of fifty children aged fifty months and under, who died of various diseases; he concludes that all inflammatory affections have a tendency to increase the weight of the organ, and that this increase is mainly due to the greater amount of blood contained in the cerebral vessels.

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* "Die Krankheiten des Gehirns und Rückenmarks bei Kindern," von L. W. Mauthner, p. 189. Wien, 1844.

Rokitansky has observed hypertrophy of the medulla oblongata, while Virchow and others have described cases of the new formation of brain substance in the form of a tumour on the walls of the ventricles.

Hernia Cerebri, or *Fungus Cerebri.*—As a result of local injury, we meet with a species of fungoid growth of the brain, which has been termed hernia cerebri, but which is very different from the hernia cerebri to which allusion has been made, in the section treating of congenital hydrocephalus. In consequence of a fracture of the skull, the brain appears to sprout forth in the shape of a vascular, medullary growth. It appears as if the brain, released from its confinement, luxuriated in its newly-acquired liberty. Extensive suppuration is generally found accompanying this form of hernia, within the brain.

According to Förster, the mass much resembles granulations on the skin, being composed of loops of dilated and elongated capillaries, from which proceed offshoots of newly-formed vessels, the whole being surrounded by granular masses and cells, having the general character of pus-cells; while the nervous structures disappear. The whole is thus a loose kind of granulation tissue, which must be regarded as an outgrowth of the non-nervous, *i.e.*, vascular and connective-tissue elements of the brain substance.

ATROPHY.

The converse of hypertrophy is a condition which, as we have already had occasion to remark, is the result of a natural process in old age; but it is also the effect of disease; or of an arrest of development. In consequence of long-standing exhausting illness in children, Dr. West informs us that the brain is found far from filling up the cavity of the skull, so that a knife may be passed in many places between it and the cranial walls. The same appearance is met with in the adult, and the consequence is that serum is effused between the brain and its envelopes in order to supply the defect; the convolutions become thinner and they are separated by broader sulci. There is some discoloration of the tissue, and the veins of the pia mater are observed to present a varicose appearance, owing to the loss of support which they experience. We also meet with partial atrophy; this is generally of a secondary character, owing to pressure exerted upon individual parts by tumours or other adventitious growths, apoplectic cysts in the arachnoid or peripheral lesions. The absorption induced by the effect of pressure is accompanied by induration of the adjoining layers of the cerebral tissue. The atrophy that is found as a result of arrest in the functions of a peripheral nerve, as in the case of the optic thalamus in amaurosis is a marked instance of the influence of functional derangement upon nutrition, propagated to a distance.

In idiots we have congenital atrophy of the entire or of portions of the brain; in the brain of old insane persons it is common, according to Neumann's statement, who has examined fifty cases of the kind, to find the posterior lobes and their convolutions more atrophied than other parts; Sir Charles Bell, Cruveilhier, and Lallemand record cases of epilepsy and hemiplegia, accompanying congenital atrophy of one hemisphere, though not necessarily associated with a destruction of the intellectual powers. Atrophy of the brain is often seen at the autopsies of persons who have died of starvation or in an extremely cachectic state; and being necessarily accompanied by a large increase of fluid within, this fact has led to such cases being described as cases of "serous apoplexy," a condition not proved to exist, and which can never be admitted as a cause of death.

Atrophy of particular parts, *e.g.*, of the cerebellum, has been known to occur.* In one remarkable case, recorded by Dr. Clapton, this portion of the brain was reduced to one-third its natural size, weighing 710 grains, instead of 2,200, which was reckoned as the mean weight. No cause could be traced.

Clapton: "Trans. Path. Soc.," 1871, vol. xxii. p. 20, Plate ii.

CHAPTER XI.

THE BRAIN—MORBID GROWTHS.

THE products of those perverted states of nutrition which give rise to new growths, are found in the brain, as in other tissues of the body, though for the most part in a secondary form; associated with or following upon their deposit elsewhere. They have certain features in common, owing to the anatomical relations of the brain; thus they all generally assume a rounded shape owing to the uniform pressure to which they are subjected on all sides; they may not proceed to a high degree of development from the early danger to life which they induce; they are liable to produce softening of the tissues in the immediate vicinity, and, owing to the impairment of the circulation, are commonly accompanied by an effusion into the ventricles. The last two circumstances are those to which we may probably refer the symptoms observed during life; for the presence of tumours in the brain is often not discovered until the death of the patient from disease of some other organ, and where they had maintained this quiescent state, the cerebral tissue in the vicinity exhibits no traces of degeneration. Thus Messrs. Tonnellé,* Léveillé, and others, who have devoted especial attention to the occurrence of cerebral tubercle, conclude that the tubercles in themselves do not give rise to any symptoms, but that the cerebral symptoms accompanying them are exclusively due to the intercurrent inflammation. The difficulty of early diagnosis, and the fact of adventitious growths in the brain occurring almost exclusively in the secondary form, place them more especially in the range of the morbid anatomist; they are even less amenable to therapeutic treatment than when they have found a nidus in the abdominal or thoracic organs. The forms which we most frequently meet with are tubercle, cancer, and a special form of tumour, glioma; but sarcoma, myxoma and other forms are occasionally seen syphilitic growths very rarely.

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* See Rilliet and Barthez: "Traité Clinique des Maladies des Enfants," tom. xiii. p. 552, et seq.

TUBERCLE OF THE BRAIN.

Tubercle is a frequent concomitant or source of meningeal inflammation. Tubercular deposit in the cerebral tissue, like tubercular meningitis, is a disease specially incident to childhood; but the two are not necessarily associated together. Tubercle in the brain may affect any part of the organ; it occurs in the shape of rounded nodes varying in size from a pin's head to a walnut or hen's egg; the deposits are not generally numerous, and their size bears an inverse ratio to their frequency. It is most common to find only one or two, and of an average size of a chestnut. Dr. Baly has recorded a case in the reports of the Pathological Society[*] of a young man who died in the Millbank prison, and in whose brain tubercles were found; only two were discovered in the left hemisphere, but those in the right are estimated to have been as many as fifty, varying in size from a grain of pearl barley to that of a barley-corn; the same case is also instructive as showing the great rapidity with which the deposit may occasionally take place under circumstances favouring the disease; for the patient was admitted into the prison on the 30th December as a healthy subject; after a few days was attacked with headache, and on the 19th of January following he died with all the symptoms of an acute cerebral affection. The case appears to disprove the dictum of Rokitansky, that cerebral tubercle never occurs in any but the chronic form.

Cerebral tubercle usually occurs in the yellow variety, presenting the appearance of soft yellow cheese, and the miliary granulations, which are so characteristic of meningeal tubercle, are very rarely met with. When they occur it is usually around a caseous mass, and hence the question has been raised whether the whole mass was originally in the condition of miliary granulations. Rokitansky is of opinion that tubercle in the brain, does, in part at least, commence in the grey translucent form, considering that portions of a tubercular mass are sometimes found in that state. In any case, however, he adds, it may continue for a short period only in that form, and soon pass into the stage of the yellow cheesy tubercle. Other pathologists would look upon the miliary granulations as the result of caseous metamorphosis in the primary mass, which thus acts as a focus of local infection. Sometimes the mass is evidently made up of an aggregation of minute granulations, and in these cases the view of Rokitansky seems very probable. In other cases, according to Förster, the transition to healthy tissue is formed by a homogeneous zone of a greyish-red colour made up of newly-formed tubercular elements; moreover the half or even the whole of the mass may be made up of such elements exclusively; showing that it is not composed of an aggregation of minute growths, but was one homogeneous mass from the beginning. This fact is remarkable, as showing that pure tubercular growth may, in the brain, attain a size far exceeding that of tubercles in any other organ. The minute structure of the more recent portions of cerebral tubercle resembles that of recent tubercle in general. They are composed of nuclei, small cells, and occasional larger many-nucleated cells. In the more central portions are seen these elements in a state of atrophy or decay (tubercle corpuscles), and the more homogeneous amorphous granular mass called caseous tubercle. These gradations of structure will distinguish tubercle from all other new growths, except the syphilitic, which it extremely resembles, and the history and concomitant morbid appearances will sometimes be the only criteria for drawing a distinction between them. If, as occasionally happens, the tubercular matter proceeds to the stage of softening, the superficial observer may mistake the morbid appearances for those of a simple abscess of the brain.

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[*] Session 1850-51, p. 34.

Cerebral tubercle is far more common in children than in adults. Out of 472 tubercular children (putting together the cases of Barthez and Rilliet, and of Dr. Hillier), eighty-seven had tubercle in the brain. Physicians who have had the most extensive experience in these matters agree as to the rarity of its occurrence in the adult; thus Cruveilhier never met with a single case, and Lugol, in the large hospital of St. Louis, has only seen eight instances, in none of which any symptoms of the disease were manifested during life. In 117 cases of adults dying of tubercle reported by Louis, it occurred only once.

It is a singular fact, which we gather from the statistics of MM. Rilliet and Barthez, that sex appears to exert a marked influence upon the occurrence of cerebral tubercle; in each variety, the males are considerably more liable to the affection than females; of forty-four cases, we find twenty-nine occurring in boys, and fifteen in girls. The fact is confirmed by the statistics of our own medical writers: an analysis of fifteen cases reported by Dr. Abercrombie and others, establishes a similar proportion; ten of these cases were males, and five females. It is however, right to state that Dr. Hennis Green's* statistics contradict this fact; his observations were made at the same hospital as those of MM. Rilliet and Barthez, and of the thirty cases which he has collected fourteen occurred in boys and sixteen in girls. Out of twelve cases described by Dr. Hillier, nine were in boys and three in girls.

The deposit of tubercular matter sometimes occurs in patches of irregular shape and size, on the surface of the brain, beneath the pia mater,† but commonly, as we have already seen, it forms nodules within the cerebral tissue. It is often met with both in cerebrum and cerebellum at the same time; the number of cases in which it occurs in one or the other alone is about equal; the pons Varolii is, in rare cases, the only seat of the deposit.

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* "Medico-Chir. Trans.," vol. xxv. p. 192.
† See Mr. Dunn's case, Ibid. p. 209.

In the lungs we frequently meet with satisfactory evidence of the power of the system to reject and cure tubercle; we are not possessed of similar proof with regard to the brain; the only analogous process is that in which the vitality of the deposit seems utterly destroyed, and cretification results; this is a metamorphosis which sometimes, though rarely, takes place in cerebral tubercle. We must not confound with cretification of tubercle, certain gritty or sabulous masses found in the brain; thus, our notes contain the history of a case, in which, on a vertical section of the cerebellum, the knife grated upon some calculous formations, imbedded in the tissue, and intimately adherent to it; there proved to be, on each side, three or four irregularly crystallized masses, which broke up easily on pressure, and were not affected by either liquor potassæ or acetic acid. There was no other perceptible disease of the cerebral tissue, but the choroid plexuses were covered with concentric corpuscles. Andral* gives, as a great curiosity, an analogous case, in which, however, the "ossifications" were enclosed in a cyst.

It should be mentioned that while miliary tubercle of the meninges is often found in the vicinity of a cerebral tubercle, there is also a form of miliary granulation found in cases of general tuberculosis in the cerebral substance under the meninges, which in its relations to the vessels and other characters precisely resembles that met with on the pia mater.

SYPHILITIC TUMOURS.

Syphilitic growths in the brain are not common. They resemble generally the gummata of other parts, being composed chiefly of greyish semi-translucent matter, which is liable to become opaque and crumbling on undergoing a caseous transformation. According to Virchow† they stand in an intermediate position between the soft gummata of mucous membranes or the gelatinous of the periosteum on the one hand, and the hard gummata of the liver and testicle on the other hand. They present, as a rule, a soft, transparent, sometimes gelatinous mass, surrounded by a delicate granulation-tissue of greyish appearance, traversed by numerous vessels, some of new formation, which gradually loses itself in the surrounding cerebral substance. In the middle there often occur limited spots of caseous degeneration, which may be two, three, or six lines thick, and are sometimes fused together so as to form one mass of irregular outline.

The caseous portions are firmer and more difficult to cut than the surrounding mass, especially if, as often happens, the latter have undergone some softening. Sometimes on the other hand, the external parts acquire a very considerable hardness, and a sort of firm, fibrous capsule is formed, enclosing the caseous masses. As to their minute structure, the younger and softer parts are principally composed of cells, usually round, with rather large nuclei, imbedded in a scanty, granular or sometimes fibrillated intercellular substance. When the fibrous element predominates, the cells are rather spindle-shaped or stellate. The degenerated portions show an imperfect, fatty metamorphosis, being composed of amorphous material derived from atrophied cells, and punctated with fatty molecules. This description (which we have taken from Virchow) obviously agrees in many points with that of cerebral tubercle, and the distinction is, as has been said, often very difficult. According to the same authority, the tubercular mass is usually more perfectly round than the syphilitic, which either has the form of the part in which it occurs, or else has a very irregular outline. Further, the larger portion of the tubercle is usually compound of caseous material, the younger and undegenerated portions being so inconspicuous as to be often overlooked, while the syphilitic gummata consists in great proportion either of fresh granulation-tissue or of a fibrous mass, the caseous portions being inconsiderable, and they never soften in the centre. It is also difficult to distinguish syphilitic growths from some forms of sarcoma and glioma in the condition of fatty degeneration.

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* "Clinique Médicale," t. v. 719.

† "Die Krankhaften Geschwülste," vol. ii. p. 454.

It should be remembered that syphilis is very liable to give rise, beside its peculiar or specific products, to other morbid formations which are not distinguishable from the products of ordinary inflammation or hyperplastic growth. Thus in the case of a syphilitic growth connected with the pons Varolii, recorded by Mr. Robinson, the report of Mr. Hulke on its minute anatomy, states that histologically it was not distinguishable from a glioma.*

CANCER.

Next in frequency to tubercular deposit we find the various forms of cancer, all of which, excepting the epithelial variety, are met with in the brain. There are no symptoms peculiar to the disease, beyond the effects resulting from pressure; and even they do not appear to be in any way commensurate with the size of the deposit. The form which it assumes is that of infiltration, without any definite limits, or of a tumour surrounded by a cyst; in the former case, there seems to be a complete replacement of cerebral tissue by cancerous growth. In other cases capillary hæmorrhage and softening, or sometimes induration of the brain substance is observed around the tumour. The size of the growths varies;

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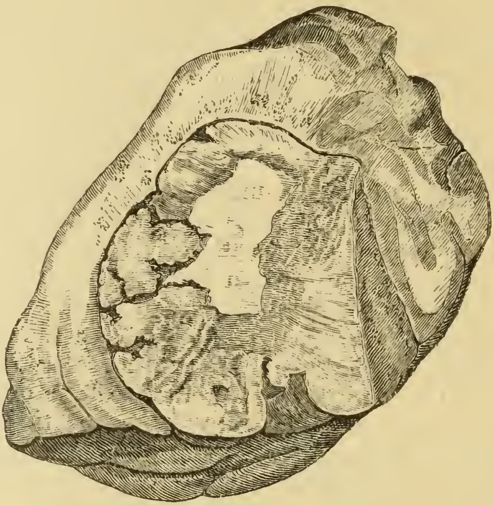
* "Trans. Path. Soc.," 1869, vol. xx. pp. 17 and 21.

they may be as large as a fist, and several are sometimes present at once. Cerebral cancer may be either primary or secondary. It occurs more frequently in the cerebral hemispheres than in other parts of the brain; thus, in forty-three cases of cancer of the nervous centres alluded to by Andral,* we find thirty-one occurring in the cerebral hemispheres, and five in the cerebellum; the remainder were thus distributed: three were found in the pituitary gland, one in the corpus callosum, and three in the spinal cord.

FIG. 68.



Cancerous tumour, occupying the upper portion of the posterior cerebral lobe of a man aged fifty-four, brought into St. Mary's Hospital, comatose and hemiplegic, in which state he remained until death. The central portion was dense and fibrous, of a yellow colour, and consisting of fusiform, fibre cells; the external portion soft, cream-coloured, or pink, composed of a variety of compound cancer-corpuscles. The whole was surrounded by a red vascular margin.

Melanotic Cancer.—The melanotic growths met with in the brain seem to be always strictly melanotic cancer or sarcoma; the simple pigmented growths seen on the membranes not occurring in the cerebral substance. The melanotic cancer is not common, and always seems to occur simultaneously with similar growths elsewhere. Sir Robert Carswell † gives a specimen of two tumours of this description, which were located in the right hemisphere of the cerebrum of a man; they were of the size of a hen's egg, and penetrated into the ventricles. Melanotic deposits were also found in other organs of the same subject, and the veins passing from the tumours in the brain were observed to contain melanotic matter in a fluid condition. Dr. Hooper's work on the brain also contains a plate representing this disease; and Dr. Clendinning brought a case of the kind before the notice of the Pathological Society.*

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* "Clinique Méd." T. v., p. 633.

† "Pathological Anatomy," 1838, Art. "Melanoma," pl. ii.

SARCOMA

Sarcomatous tumours of the brain have the same general characters as these tumours have elsewhere. Virchow† distinguishes two forms of cerebral sarcomata—the hard and the soft. The former are masses of dense homogeneous growth, very slightly vascular, and clearly marked off from the surrounding tissue by a more vascular zone; so that they can be very easily detached. In their minute structure they are composed of spindle-shaped cells arranged in a parallel manner, with a very distinct, firm, sometimes fibrillated intercellular substance. The soft variety sometimes offers forms which constitute a transition to a myxoma or glioma, but are distinguished from these growths by the great abundance of cells, which so preponderate as to give the tumour a medullary appearance; and there is no doubt that many tumours described as medullary cancer of the brain would by modern systematists be referred to this group.‡ This form of sarcoma is described as of a greyish rather than a white colour, and usually very plentifully supplied with blood-vessels. It may, however, undergo fatty degeneration, becoming yellow or opaque. The cells may present all the varieties which have been described as incident to the cells of sarcoma, but spindle-shaped cells, with remarkably long processes and small cell-body, are especially noticeable. The intercellular substance is scanty and inconspicuous. Portions of the tumour may sometimes be transformed into cysts, but without a distinct lining membrane. Hæmorrhagic changes are not common. Cerebral sarcoma is always single; it does not generally spread by continuous infection; nor has it ever been known to give rise to secondary tumours in other parts. On the other hand, sarcoma of the brain may arise as a secondary growth, consequent on tumours of the same kind elsewhere. A remarkable example of this mode of origin is recorded in the Transactions of the Pathological Society for 1867 and 1868; where a spindle-celled sarcoma of the head of the tibia, which had existed for many years, was removed by amputation, and seemed to be completely cured. A year after the operation, cerebral symptoms supervened, and the patient died with numerous tumours in the brain, partly connected with the pia mater, partly isolated, which had the same type of structure as the primary tumour of the tibia.§

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* "Report of the Pathological Society of London," 1847, p. 15.

† "Die Krankhaften Geschwülste," ii. 377.

‡ Thus a tumour described by Messrs. Gay and Toynbee in "Trans. Path. Soc.," 1858, vol. iv. p. 16, pl. i. fig. 1 would undoubtedly now be called a sarcoma.

§ "Trans. Path. Soc." vol. xviii. p. 215, pl. iv. figs. 4, 5, 6, and vol. xix. p. 33.

GLIOMA.

This name has been given to a new growth arising in the special fundamental substance or cement of the brain, which supports and unites the true nervous structures. This fundamental substance, the existence of which has only lately been recognized, is composed mostly of soft, finely granular material, in which are contained small cells, mostly round, and situated at a certain distance from one another. Sometimes the cells appear to be connected into a fine reticulation, but it is not quite certain that this appearance is not due to certain methods of preparation. To the whole structure Virchow has given the name of neuroglia (nerve-cement), and histologists are now agreed as to its existence, though not with respect to all its characters. A general increase or hyperplasia of the neuroglia constitutes, as we have said, so-called hypertrophy of the brain; its partial increase in the form of a tumour, produces glioma. Since the neuroglia is, in fact, a kind of connective tissue, it is natural that its special developments should have a great resemblance to special developments of connective tissue in general; and this is found to be actually the case, since it is doubtful whether gliomata can be strictly separated from the group of sarcomata. The former differ only in representing a more special type of structure; while a sarcoma reproduces the embryonic or rudimentary condition of connective tissue in general.

Glioma, when examined fresh, may seem to consist merely of a homogeneous basis substance, with nuclei, but by hardening or sometimes at once, cells are seen which are $\cdot006^{\mathrm{mm}}$ to $\cdot012^{\mathrm{mm}}$ [$\frac{1}{4000}$inch to $\frac{1}{2000}$inch] in diameter. They are seen in hardened specimens to be imbedded in an intercellular substance like that of the normal neuroglia (see Fig. 35, p. 172) which is subject to great variation, both in amount and character. The consistency of the tumours is also variable; the harder forms approach fibrous tumours; the softer approximate to the structure of myxoma. They are usually well supplied with vessels, but are liable to undergo fatty or caseous degeneration in the centre. They are quite continuous with the brain-substance all round. Cerebral gliomata generally occur as solitary tumours; they do not affect the membranes or neighbouring organs, or give rise to secondary formations elsewhere; they are, in fact, not at all malignant. The parts of the brain chiefly affected are the cerebral hemispheres, and especially their posterior lobes; the harder forms occur on the walls of the ventricles, the softer within the substance of the hemispheres. They are very rare in the central ganglia of the brain, tumours in this situation being mostly sarcomata or cancers.

We may mention that these tumours occur also in the spinal cord, along the cranial nerves, and in the retina. In the latter situation they appear to be often locally infectious and malignant.

Glioma[*] often approaches in structure to a sarcoma or a myxoma; and mixed forms are not uncommon.

Fatty Tumours.—The simple fatty tumour is only found in the choroid plexus, where it does not, however, attain any great size. The fatty growths most frequently met with in the brain are those which are termed cholesteatoma; they are formed of concentric layers, and present a metallic lustre; they consist of membranous layers, ordinary fat vesicles, and cholesterine plates, and are inclosed in a capsule. They attain the size of a walnut, or goose's egg. These tumours most commonly arise from the membranes of the brain, but may originate in the cerebral substance itself.

Fibrous Tumours are, according to Förster, extremely rare; they are usually single, and form clearly defined nodules, the size of a pea or a hazel nut, rarely larger.

Myxoma.—This form of tumour is, according to Virchow,[†] occasionally met with in the brain, and especially in the cerebral hemispheres. It forms very soft gelatinous masses, which frequently break down into cysts containing a mucous fluid. They may attain the size of a man's fist.

CYSTS.

Cysts of various kinds present themselves in the brain; those resulting from apoplectic effusions are the most common, and present, as we have seen, various stages of development. It is probable, that in many instances, their formation may be due to the same process as that described by Mr. Prescott Hewett, as giving rise to the inter-arachnoid cysts, viz., a formation of a false membrane, subsequent to the effusion from the sanguineous clot. These cysts, it should be remembered, may ultimately come to contain a colourless serous fluid. They are characterized by the firmness and thickness of their walls. Abscess and softening may equally give rise to cysts.

Echinococcus or *Hydatid* cysts are also found. They occupy the peripheral more frequently than the central portions of the hemispheres, and are found to present no connection with the surrounding tissues. They are, according to Dr. Bastian, almost always barren, and thus correspond to the acephalocysts of Laennec. When met with in the brain there is generally a coincident development of the same parasites in the liver, a fact first pointed out by Aran,[‡] who has analyzed forty-seven cases of this kind. They are mostly met with in young persons from ten to twenty years of age.[§]

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[*] Cases of glioma of the brain are recorded by Dr. Cayley. in the "Trans. Path. Soc.," 1865, vol. xvi. 23; Dr. Moxon, Ibid. 1868, vol. xix. 21, pl. x. fig. 3; and by Dr. Green, Ibid. 1869, vol. xx. 24.

[†] "Die Krankhaften Geschwülste," ii. 423.

[‡] Schmidt's "Jahrbücher der Medicin," vol. xxxiii. p. 136.

[§] Further very copious references are given by Dr. Bastian in Reynolds's "System of Medicine," vol. ii. p. 498.

Cysticercus cellulosœ is either confined to the brain or else there is a simultaneous development in the muscles. It may occur in very large numbers; according to Cobbold, at least 100 fatal cases have come to light.

Serous cysts, neither originating in hæmorrhage nor due to parasites, are of very rare occurrence; but true *Dermoid* cysts have been recorded. In a case referred to by Förster a compound cyst, containing hair, cartilage, and bone, was found in the left cerebral hemisphere of a boy ten weeks old. Another containing fat, with short stiff hairs, was seen by Sir J. Paget, under the cerebellum of an elderly man.*

CONDITION OF THE BRAIN IN INSANITY.

Although there are no specific morbid conditions always associated with insanity some facts have been ascertained which are worth consideration. They must necessarily be given in a somewhat fragmentary form. In acute mania we find evidences of great hyperæmia, or incipient inflammation, especially in the cortex of the brain.† The grey matter (and especially its inner two-thirds) assumes a distinct pink tint, which is not removed by washing the cut surface. The same is seen in a less degree in some parts of the white medullary substance. This intense hyperæmia is probably always accompanied by some degree of capillary hæmorrhage; and on microscopical examination red blood corpuscles may be seen scattered among the nervous elements. To this condition especially belong the so-called dissecting aneurisms; though we must repeat that they are not peculiar to cases of insanity. A more chronic change consists in an increase of connective tissue, by which the nervous elements are compressed and impaired. This connective tissue growth appears to start from the walls of the capillary vessels themselves, and not from the neuroglia. The vessels suffer also more obvious changes; they become varicose, tortuous, knotted, or aneurismal; their walls show great thickening and fatty or calcareous degeneration. These changes in the vessels are especially characteristic of insanity, accompanied by general paralysis.‡ Degeneration of other structures is shown by the presence of amyloid corpuscles, pigmented, fatty, and atrophied nerve cells. Actual calcification of the ganglionic cells has also been observed. Peculiar changes in the nerve cells have further been observed by Meynert, who describes vesicular enlargement or division of their nuclei, œdema, and hardening, accompanied by enlargement of the cells themselves, with final molecular decay, and atrophy.*

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* "Surgical Pathology," second edition, p. 435.

† We believe this form of hyperæmia to be essentially different from that caused by meningitis. It is distinguished by the stratified appearance of the cortex, and by the red tint being chiefly seen in the inner part, while the outer layer is pale. In meningitis, or simple congestion, on the other hand, the whole of the grey cortical substance has a uniform pinkish tint.

‡ Described by Rokitansky, Wedl, Sankey, and others. See description and figures by Sankey in "Trans. Path. Soc.," 1866, vol. xvii. pl. i. p. 8.

Drs. Tuke and Rutherford,† in an analysis of the morbid appearances in the brains of thirty insane persons, found in eighteen cases increase of connective tissue (grey degeneration or sclerosis), which they regard as proceeding from the neuroglia. They also describe the occurrence of cavities sometimes as much as $\frac{1}{50}$inch in diameter.

Changes in the Brain in General Paralysis.—One form of insanity, known as general paralysis of the insane, has been made the subject of a very large number of pathologico-anatomical investigations, and it has often been thought that some lesion had been discovered which could account for the disease.

Adhesion of the meninges to the cortical substance of the brain was first thought to be the clearest pathognomonic sign of the disease; then softening of the cortical substance, and its easy separation, or splitting into layers, was regarded as the criterion; induration of the white substance also had its defenders; and other observers attached very great importance to a granular and sandy condition of the inner surface of the cerebral ventricles. There is, however, no one of these characters which is not met with in morbid conditions of the brain, associated with very different diseases. Certain morbid conditions, supposed to occur in the cerebellum, are even less constant and important. When the microscope came to be applied to the investigation of the brain other changes began to be signalized. Rokitansky was the first to draw attention to an increase in the connective tissue or neuroglia, especially of the cortex; and his observations have been in the main confirmed by others who have used very different methods of preparation. The change is not, however, confined to the brain; it is, on the other hand, found in a more advanced stage in the cord, and appears to extend itself by ascending progress to the brain. It is probable that when not widely distributed, or arrived at a high degree of evolution, this change may produce hardly any symptoms. The order in which it attacks first the spine, then the brain, accords very well with the symptoms of general paralysis. Beside this chronic increase of the neuroglia, certain changes in the nervous elements have been observed; they are described by Dr. Lockhart Clarke as unduly pigmented; but this phenomenon does not appear to be constant. In one case examined by Dr. Clarke,‡ he notices the occurrence of small cavities in all parts of the brain, some symmetrical and smooth, others with ragged edges. They appear to correspond precisely with what have been seen by several observers, and very variously interpreted. They were first described some years ago by Durand Fardel,* in what he terms the "état criblé" of the brain; a condition observed by him in the brains of old persons, most of whom suffered from delirium or mania, combined with paralysis. Durand Fardel himself attributed these phenomena simply to congestion, causing dilatation of the cerebral vessels, and consequent destruction of the surrounding brain substance; and he attached no importance to them. Laborde,† on the other hand, has observed such cavities, which he calls "lacunes pisiformes," and believes them to be connected with phenomena of diffused general paralysis in the aged. It thus seems that though they have no special connection with psychical manifestations, they are, at all events, frequently seen in diffused cerebral paralysis, as well as in general paralysis, properly so called. Many of the cases in which such lesions were observed by Messrs. Tuke and Rutherford, were accompanied at least by paralysis. They may very reasonably account for local paralysis, or other affections of nerve function; and it is probably in this way that they are of importance in the disease now to be considered.‡

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* "Vierteljahrsschrift für Psychiatrie," 1868. Heft 3 and 4; quoted in "Jahres bericht der Medicin für 1868," vol. ii. p. 19

† "Edin. Med. Journ.," Oct., 1869, p. 289.

‡ "Journal of Mental Science," Jan., 1870.

CHANGES IN THE BRAIN IN DIABETES.

Dr. Dickinson§ has lately described with much care certain conditions of the nervous centres in patients affected with diabetes. The parts chiefly noticed were the pons Varolii, the medulla oblongata, and the cerebral ganglia, as well as the spinal chord; but the cerebral hemispheres were examined in two instances. In all these parts were seen small cavities, varying from $\frac{1}{140}$ inch in diameter to the size of a small pea, containing "débris of cerebral tissue" and yellow pigment. These cavities in most cases obviously, and probably in all, surrounded small arteries (which were themselves dilated), and many of them formed channels of which the width was very small in proportion to the length. In what seemed to be the earlier stages of the morbid process, there was simple dilatation of the vessels with surrounding hæmorrhage, but without any obvious rupture. Similar changes were seen in the spinal chord. Seven cases were examined, and the appearances in all showed a substantial agreement. The nerve cells are described as healthy. It is, we think, obvious that the changes described by Dr. Dickinson in their slighter degrees are not different from those which have been observed in various morbid states of the brain referred to above. When he describes (Case 4) the white matter of the cerebral hemispheres as "full of holes, of which the larger were $\frac{1}{20}$ inch across," and which "lay so close together as to give a cribriform appearance to the section, the condition is strikingly like the "état criblé" first described by Durand Fardel, and now believed to be owing to atrophy of the brain substance, with subsequent or antecedent dilatation of the perivascular lymphatic sheaths of the cerebral arteries. The cases (4 and 7) where blood globules and yellow pigment arising from degenerated blood were found around the vessels, are evidently instances of that hæmorrhage into the perivascular sheaths, which is probably of very common occurrence. In the extreme cases there was evidently destruction of nervous tissue, and the changes are such as have apparently not been elsewhere described, except in connection with general paralysis of the insane; and the fact that they were especially noticeable in the pons and medulla oblongata, *i.e.*, approximately in that physiological region, injury to which is known to produce diabetes, give Dr. Dickinson's researches a peculiar interest. The morbid appearances in cases of diabetes described by some French authors refer chiefly to the nerve cells, and were neither so well marked nor so clearly described. They are, however, more definitely assigned to the "diabetic region."

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* "Maladies des Vieillards." Paris, 1854, p. 51.

† "Ramollissement et Congestion du Cerveau," p. 94. Paris, 1866.

‡ "On General Paralysis." See Rokitansky, "Ueber Bindegewebswucherung im Nerven system," 1857; Lockhart Clarke, "Lancet," Sept. 1st, 1866; Magnan, "Archives de Physiologie," 1868, vol. i. p. 322; Maudsley, Reynolds's "System of Medicine," vol. ii. p. 50. The two last-named writers give many references.

§ "Medico-Chirurgical Transactions," 1870, vol. liii. p. 233, pl. vii. and viii.

THE PITUITARY BODY.

The pituitary body presents morbid conditions which, generally, are rather pathological curiosities, than that they offer any peculiar points of general interest; in a physiological point of view, tumours or cysts occurring in it attract attention, from their not producing those symptoms which are generally attributed to pressure upon the encephalon, and this is supposed to be due to the force acting in an upward direction. Thus they are rarely accompanied by paralysis, though acquiring an extent sufficient to displace the lateral ventricles, with the thalami and corpora striata, a circumstance presenting, as Dr. Romberg remarks, an analogy to the different effects produced upon the conduction of a nerve by a tumour, according as the nerve is gradually distended or forcibly compressed. Neither Rokitansky, who treats diseases of the pituitary body in detail, nor Engel, who has written a monograph on the subject, corroborates the observations of Joseph Wenzel, that disease of the pituitary body is an essential feature in epilepsy. The pathologists of our own country have not observed a relation of the kind. Epilepsy is met with as a result of the most various degenerations, or morbid products within the brain, independently of any marked disease of this appendix cerebri; it is not constantly associated with any one lesion, and in the cases of disease of the pituitary gland, given, for instance, by Dr. Bright,* we find no epileptic seizures during life, and conversely, we see epilepsy occurring without any disorganization of this part.

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* Reports, &c., case cxlii.

The pituitary body does not seem to bear any definite relation to the manifestations of the mind. Dr. Bright * gives an instance in which it was absent; the patient was a man who died at the age of forty-eight, of softening of the left corpus striatum; but he had enjoyed thorough good health until five months previously.

In the course of our account of cerebral morbid anatomy, we have had occasion to allude to the occurrence of aneurism of the cerebral arteries; the subject will meet with a fuller consideration when we treat of the diseases of the vascular system, but we could not admit the pathology of the brain without pointedly remarking upon their importance in the production of cerebral symptoms, and as a more or less direct cause of death. The diseases of the arteries play a most important part in the production of cerebral disease, and in many of the morbid conditions which we have passed in review they may be viewed as one of the main elements.

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* An interesting case of disease of the infundibulum and pituitary body is given in the "Records of the Pathological Society of London" for 1849, p. 19.

CHAPTER XII.

THE SPINAL CORD AND ITS MEMBRANES.

We must commence this section with the ungratifying confession, that it is a subject upon which our knowledge is very limited. At present, we can scarcely be said to have advanced beyond the very threshold of this department of science.

We shall follow the same order we adopted in treating of the morbid anatomy of the brain, and examine successively the post-mortem appearances of the dura mater, the arachnoid, and pia mater, and then of the cord itself. In all cadaveric examinations of the spinal column, it is particularly to be borne in mind, that the position of the body after death may influence the post-mortem phenomena, independently of morbid action, owing to the gravitation of the fluids to the depending portions, and their secondary effect upon the nerve tissues. How important it is to attend to this point is illustrated by some observations, made by Mr. Curling,* of tetanic cases. On examining the body of a man who had died of tetanus, which had been placed on its face immediately after death, Mr. Curling found that part of the pia mater covering the anterior columns of the medulla spinalis remarkably vascular; a circumstance which would necessarily induce a conviction in the mind of the pathologist, that an essential lesion had been discovered, by which the exaltation of motor action could be satisfactorily explained. Unfortunately for the conclusive force of the observation, in three other instances, where the bodies were suffered to remain in the usual position, the vessels on the posterior parts only were observed to be turgid.

This influence of position is more likely to affect the spinal cord than the brain, owing to its being less excluded from atmospheric agency. It is also important to remember, that the relation of the envelopes of the cord differs from that existing between the investments of the brain and their contents in various material points. The movements of the osseous case of the cord would have rendered a close adhesion with the membranes a source of frequent danger; we may fairly assume this as a reason why the dura mater of the spinal column is only very loosely attached to the vertebral canal; on which account it allows of an accumulation of fluid on its external surface, such as we but rarely meet with in the brain. Owing to the firm attachment of the dura mater to the occipital foramen, fluids accumulated at this part are prevented passing into the cranial cavity, while there is a free communication between the arachnoidal spaces of the two cavities. This fact is one that must not be overlooked in morbid affections both of the spine and the encephalon; independent of the protection that the arachnoidal fluid affords to the cord, it is an evident means of securing a balance in the circulation in the nervous centres, while, on the other hand, a derangement in its quantity and site may be alone sufficient to produce serious symptoms, which the morbid anatomist would be unable to measure by physical tests.

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* On "Tetanus," p. 48.

Spinal Dura Mater.—We possess no evidence of the occurrence of idiopathic disease of the spinal dura mater, though it can scarcely be supposed that a fibrous membrane, situated as it is, should not suffer from the rheumatic diathesis. In all cases of injury of the vertebral column it is liable to be affected, and scrofulous disease of the vertebræ, and the extension of psoas abscess, may involve it.

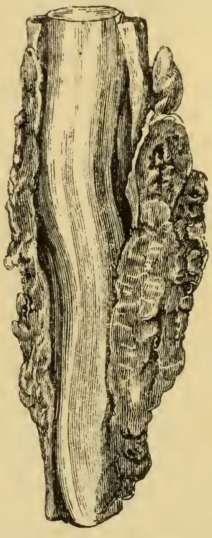
Spina Bifida.—In that congenital affection resulting from deficiency of the posterior arches of some of the vertebræ, called spina bifida, which is analogous to the hydrencephalocele, occurring in infants, as a result of non-closure of the cranial bones, the dura mater extends into the cyst that shows on the dorsal surface of the column; but it is occasionally found deficient at one point, so that the contained fluid is only retained by the thin meninges. This pathological state differs from that to which we have compared it in this, that the fluid is entirely external to the nerve tissue; but more rarely the protruded sac contains some portion of the spinal cord itself, forming the so-called *myelocele;* which is analogous to the true encephalocele of the cranium.

The tumour varies in size from that of a small nut to that of a child's head; it is generally solitary, and occupies the lumbar or sacral region; when occurring in the back we may expect to find another tumour of the same description lower down. The swelling is of a semi-globular, or ovoid shape, and may appear pediculated, owing to a constriction at its base. The thinness of the cutaneous covering passing over the tumour, has induced some pathologists to deny its presence; this, however, is an error.

Morbid Growths.—The rarity of the occurrence of idiopathic diseases in the dura mater, applies equally to adventitious products. Encephaloid, and other forms of carcinoma, undoubtedly occur primarily in the dura mater, but in many of the instances on record, it is manifest that the disease extended from the bones to the theca vertebralis.

An instance of melanotic growth apparently proceeding from the dura mater of the cord, is to be found in the Report of the Pathological Society for 1847. Dr. Williams discovered it in a patient, aged forty-six, who, three years previously, had suffered from hemiplegia of the right side, following the extirpation of the right eye for fungus. The patient recovered from this, and in the summer preceding his death was attacked with epilepsy: weakness and numbness of the lower extremities, and inferior portion of the trunk, soon proceeding to complete paraplegia, supervened. The brain and its membranes were found healthy; within the spinal canal, closely adherent to the theca externally, there existed an irregular encephaloid mass, mottled with dark spots, extending from the third to the sixth dorsal vertebræ, the bodies of which were carious and infiltrated with cancerous matter; the portion of the cord beneath the tumour was flattened, soft, and wasted. In connection with this subject, we may also be allowed to mention the very rare occurrence of a genuine fatty tumour, within the spinal column, in contact with the dura mater; an instance of this affection was brought before the Pathological Society, in 1852, by Mr. Obré, in which death was produced in an otherwise healthy child by the mere mechanical pressure exerted by a deposit of this kind. The lipomatous growth was two and a-half inches in length, the breadth of the canal, and about half an inch in thickness, composed of the ordinary spherical fat cells: it did not differ from fat usually met with in other situations, excepting that the cells seemed to contain fat in a more solid and granular state. It lay between the theca and the bodies of the last cervical and first dorsal vertebræ. Small masses of fatty tissue in this situation are not uncommon. A tumour of somewhat uncertain histological character, growing from the arachnoid surface of dura mater of cord is described by Dr. Wilks,* and one external to dura mater by Dr. Ogle.†

Fig. 69.



Part of the dorsal portion of the spinal cord of a young man who died paraplegic.

A thick layer of lymph and tuberculous matter was found surrounding the dura mater, and slightly compressing the cord. It was manifestly an extension of disease from the adjacent vertebræ and intervertebral cartilages. The cord and dura mater appear healthy in texture.

(From St. Bartholomew's Museum, Series vii. No. 10.)

Tubercle is occasionally met with in the spinal dura mater, which thus contrasts with the cerebral; while, on the other hand, the spinal arachnoid seems to have no liability to that affection.

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* "Trans. Path. Soc.," 1856, vol. vii. p. 37.

† Ibid. p. 40.

Isolated tubercles have been seen, either independently, or associated with scrofulous disease of the vertebræ; and Dr. Moxon has described a case of miliary tubercle of the spinal dura mater associated with tubercular cerebral meningitis.*

A tumour of uncertain character from the dura mater, and compressing the cord, is described and figured by Dr. Cayley.† We should be inclined, although the reporter of the case gives it no name, to regard it as a psammoma, analogous to the growths of the cerebral dura mater which have been so called.

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* "Trans. Path. Soc.," 1870, vol. xxi. p 12.

† Ibid. 1865, vol. xvi. p. 21, woodcuts 6 and 7.

CHAPTER XIII.

THE ARACHNOID AND PIA MATER OF THE SPINAL CORD.

Although the anatomical connection between these membranes is somewhat different from that obtaining between the cerebral meninges, it does not appear that their relation in disease differs materially from what we have found to prevail in that locality; we shall, therefore, consider them together.

The absence of valves in the spinal veins and their peculiar distribution, cause the circulation of the spinal cord to be very sluggish, and therefore prone to congestion and stagnation; to this. Ollivier attributes the great number of dilatations which we find in the different points of its extent, in individuals advanced in years. He adds, that he has generally remarked that the quantity of serum in the vertebral canal was so much the larger, according as there was a greater congestion in the veins of the spine, and of the membraneous coverings of the cord; thus the slowness and difficulty of the course of the venous blood may be here the causes of a dropsy, which is independent of inflammation of the spinal membranes. These effusions of serum will, according to the exciting causes, be of a chronic or acute character; in infancy, irritation frequently gives rise to a more rapid accumulation of fluid, while in old age a slow effusion is frequently met with, which Rokitansky attributes, in part at least, to a secondary congestion, arising from atrophy of the medulla and the roots of the nerves. The fluid exhaled under such circumstances will follow the law of gravitation, and accumulate at the lower end of the spinal cord, and thus assist in exciting and perpetuating paraplegic symptoms of which we may be unable to detect a sufficiently satisfactory reason after death.

Hæmorrhage.—The occurrence of sanguineous apoplexy of the meninges, in any form, is not frequent. Dr. Abercrombie gives a single instance, which occurred under his own observation, in a child, aged seven, in whom, after an illness of three days, death ensued after violent convulsions. A long and very firm coagulum of blood was found, external to the cord, extending the whole length of the cervical portion. An interesting case of hæmorrhage under the pia mater, but external to the cord, is also quoted, from Dr. Stroud's notes, by Dr. Bright (p. 340). Numerous instances of spinal apoplexy occurring in children are given by Dr. Mauthner, but as no post-mortem appearances are recorded, the inferences are solely derived from the symptoms, which do not enable us to state positively the exact nature of the effusion. A tumour described by Dr. Bennett as a cyst compressing the cord and originating in the sub-arachnoid space, appears to have been an old apoplectic cyst.*

Spinal Meningitis.—We possess more satisfactory and copious evidence regarding the inflammatory affections of the spinal meninges, and it appears that it is a very frequent cause of death in new-born infants; thus, Billard found, that in thirty cases of convulsions, there was meningitis of the cord in twenty, only six of which presented inflammation of the cerebral meninges. It is much less frequent in the adult, and is here almost invariably associated with, or consequent upon, cerebral inflammation. As a result of an acute inflammation of the membranes, we find lymph or pus exuded, to a greater or less extent. Either may invest the entire surface of the cord, or it may be limited, as in a case that fell under our own observation, in a child four years of age, to a space of an inch and a-half in length. Cases of cerebro-spinal meningitis are, as we have before remarked, frequently epidemic, but occasionally met with sporadically. We must be careful in at once concluding, that we have to deal with a case of spinal meningitis, because we find the theca vertebralis lined with pus, for it may find its way from without into the cavity; thus, in a case of psoas abscess, given by Dr. Bright, the sudden supervention of fatal symptoms was manifestly due to this cause; a probe could be easily passed from the intervertebral foramina into the adjoining abscess. In the chronic form of spinal meningitis, the traces of the disease consist in greater or less opacity and thickening of the arachnoid, which frequently is found closely adherent to the spinal cord, and corrugated.

TETANUS, TRISMUS NEONATORUM, AND HYDROPHOBIA.

These diseases have been at different times supposed to depend upon inflammation of the spinal cord or its membranes, but as it seems without sufficient cause.

The appearances met with in *trismus neonatorum*, are:—Congestion of the spinal arachnoid, with an effusion of blood or serum into its cavity; and Dr. West† also states, that in the three cases which he examined he found effusion of fluid or coagulated blood in the cellular tissues surrounding the theca of the cord. This is not necessarily at variance with the statements of Dr. Schöller* and Dr. Colles,† who attribute trismus to inflammation of the umbilical arteries, as this may, and is, found to coexist with the former. Symptoms of congestion of the spinal membranes have also been met with in tetanus, but in by far the greater number of cases, examined after death, no uniform or adequate cause, to which the symptoms were referable, could be discovered. Observers are generally agreed that, at least in the great majority of cases, the appearance of the cord and its membranes, is to the naked eye perfectly normal. The condition of the spinal cord itself in this disease will be discussed further on.

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* "Trans. Path. Soc.," 1856, vol. vii. p. 41.

† "The Diseases of Infancy and Childhood," p. 125, 1st edition.

Hydrophobia.—The same absence of uniform pathological data exists in another disease which we cannot but refer to the nervous centres, hydrophobia ; congestion of the cerebro-spinal membranes and nerve matter, and some occasional effusion, is all that is generally met with in the shape of post-mortem effects ; we need not add that these appearances cannot be considered as characteristic of the disease in question. Mr. Youatt, whose extensive experience of hydrophobia in the brute creation justifies our referring to him as an authority, states that the appearance of inflammation of rabies is of a peculiar character in the stomach, but that no conclusion can be drawn from the state of other organs.

CHRONIC INFLAMMATION AND THICKENING.

In cases of long-standing paralysis and paraplegia, we find evidence of chronic meningitis of the cord in the corrugation, opacity, and close adhesions of the membranes to one another, and to the cord. A good illustration of this is afforded by a case given in Dr. Bright's Reports (page 380) ; here the dura mater of cord was unusually firm and thick, and, as far as the middle of the back, closely adherent to the pia mater, from which, in most parts, it could not be detached without lacerating the cord. On attentive examination it was found that the apparent thickening of the dura mater depended chiefly on a layer of membrane, of almost cartilaginous thickness, beneath it, and was, probably, rather the diseased arachnoid, or an adventitious deposit, than the dura mater itself. These appearances may be associated with further lesion of the cord, or with adventitious growths of the vertebral column or arachnoid. A remarkable case of thickening and induration of the spinal arachnoid and pia mater is recorded in the "Pathological Transactions" for 1869 (vol. xx., p. 354) by Mr. W. Adams, and reported upon by Dr. Lockhart Clarke. (See Figs. 70 and 71.)

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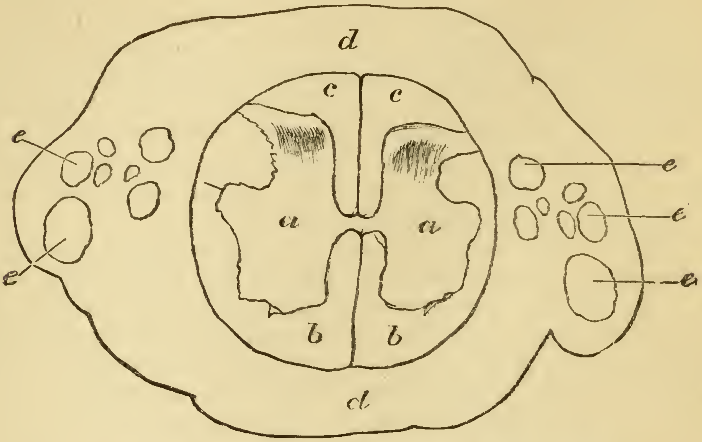
* "Neue Zeitschrift für Gebürtskunde," von Busch, D'Outrepont und Ritgen, vol. v. p. 477.

† "Dublin Hospital Reports," vol. i, p. 285.

The thickening extended nearly the whole length of the cord, but was most marked in the lumbar enlargement, where the new growth surrounded and separated the nerve roots both anterior and posterior; and the bundles forming the cauda equina. The

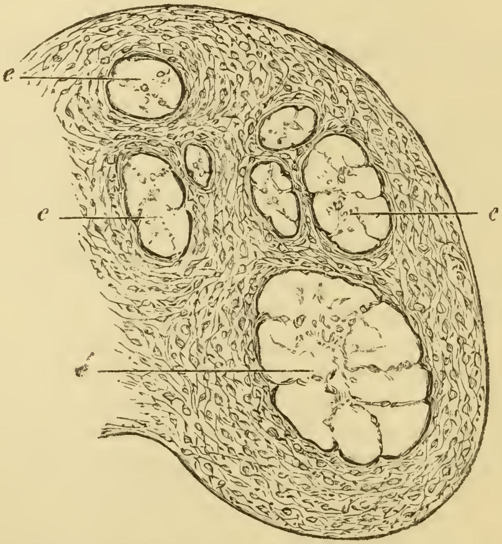
FIG. 70.



Section through the spinal cord and membranes.

a a, Cornua of grey matter; *b b*, *c c*, posterior and anterior columns; *d d*, new growth; *e e*, nerve roots involved in the new growth.

FIG. 71.



Portion of the same more highly magnified, showing the nucleated new growth of connective tissue.

e e, Nerve roots; *e'* a nerve root partly infiltrated with new growth.

("Trans. Path. Soc.," vol. xx.

structure of the new growth was found to be hypertrophied and nucleated connective tissue. It is described as being greyish and translucent.

Bony Plates in the Arachnoid.—Growths of this kind are of frequent occurrence on the visceral side of the membrane. In this respect we perceive a characteristic distinction between the head and the spinal column, for, while in the former, ossific deposits are common in the dura mater, and are scarcely ever met with in the other membranes, in the spinal column they are found to prevail in the arachnoid, and not to affect the dura mater. A remarkable instance is recorded by Herbert Mayo, in his "Outlines of Human Pathology," of osseous concretions surrounding the posterior roots of the nerves, and proceeding to the lower extremity. This undoubtedly belonged to the class of pathological products under consideration; and the case has a special interest, from its bearing upon an important law of nervous conduction. The pains suffered by the patient were so limited to the lower extremity, and were so excruciating, that the surgeon performed amputation of the limb, though with what results need scarcely be stated.

Cartilage also forms, though less frequently on the arachnoid. A good instance is recorded in the Reports of the Pathological Society of London, by Dr. Quain,* who describes the laminæ as composed of a transparent matrix, in which were deposited small cells, containing nucleoli, and numerous small amorphous granules.

Tubercle of the Spinal Meninges.—The spinal pia mater appears to be by no means so liable to be the seat of the tubercular growth as is that of the cerebrum. Rokitansky remarks on the subject, that he has never had occasion to suspect the exudation formed on the pia mater to be of a tuberculous nature. An acute tuberculosis, he adds, he has never observed in it.

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* "Reports," &c., 1849, p. 25.

CHAPTER XIV.

THE SPINAL CORD.

The forms of disease and their effects, which present themselves in the spinal cord, closely resemble those we meet with in the brain. The spinal cord does not appear to be so often attacked as the encephalon, and as we have already pointed out, it is the part which is generally left unexamined, unless attention is forcibly directed to it by the previous symptoms of the patient; for both reasons the records of its pathological states are much more scanty than those regarding the brain, and future inquirers have yet a large field to explore. The evanescent character of congestion rarely allows of its being demonstrated after death; though it is impossible to believe that there should not be accumulation of blood in the cord in those instances in which the symptoms demonstrate intense irritation of the part, as in tetanus. A case of hydrophobia is recorded by Dr. Bright, in which a blush of redness was perceived in the cineritious part of the spinal cord opposite the second and third cervical vertebræ. An anæmic condition of the cord is as difficult to demonstrate as its converse, though here too the practitioner will not fail to suggest instances in which its existence may be fairly assumed during life. Both states manifest themselves in the secondary effects of hypertrophy and atrophy. These may be general or local; the former affection belonging chiefly to early life, and the latter, like the corresponding condition in the brain, to old age.

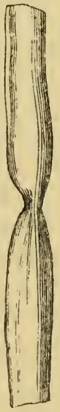
Atrophy of the spinal cord, except as the consequence of some special disease, is not very common. It is said by Rokitansky to accompany atrophy of the brain in old age; and is usually attended by some degree of induration. It also occurs in middle life, in the case of those who are compelled to pass many years in a recumbent posture; or in whom, from any cause, the lower limbs are little used. The editor has observed unmistakeable atrophy of the cord in the case of a man who had been afflicted since infancy with double talipes equinus. Atrophy of particular regions is often caused by injury, pressure of displaced vertebræ, of tumours, or inflammatory products, as seen in Fig. 72. Atrophy of particular tracts, that is of the portion of either the anterior or posterior columns connecting the brain with particular nerves, is seen as a secondary affection resulting from the functional inactivity of the nerves, or affection of that part of the brain with which they are connected; and sometimes special tracts become atrophied without any previous central or peripheral affection; but these changes will be considered under the head of degenerations.

Hypertrophy of the cord is said by Rokitansky to accompany hypertrophy of the brain in children, and to be characterized by swelling with anæmia, a rounded uniform shape, and a tough consistence. It is however, as compared with the cerebral affection, very rare.

Local enlargement of the cord is also observed above the seat of atrophy arising from pressure; but this enlargement appears, according to Förster, to depend upon inflammation.

Hyperæmia, or *congestion* of the cord, is described as an independent affection by medical writers, but it can scarcely be said to be a condition recognizable by morbid anatomy; the post-mortem appearances being, in Dr. Radcliffe's words, "very vague and unsatisfactory." The condition of the cord met with in tetanus perhaps most nearly approaches, in its naked eye characters, to what is meant by congestion, but it cannot, in reality, be simply thus defined.

FIG. 72.



Part of a spinal cord from a case of paraplegia, with angular curvature of the spine, in a lad aged eighteen.

Opposite the contracted part of the cord, a short process of bone projected from the angle of the curvature into the spinal canal.

(From St. Bartholomew's Museum, Series vii. No. 7.)

Condition of the Cord in Tetanus.—The first observations on this subject are those of Rokitansky, published in 1857. His first case is dated 1850. In this the cord was found pale, infiltrated with a greyish viscid fluid, and so far softened as to swell out in an unusual degree beyond the membranes when cut. In the viscid fluid which thus exuded were found detritus of nerve structures and irregularly swollen nerve fibres. The alteration was uniform through the whole of the cord, and showed no obvious starting point. The same appearances presented themselves in numerous similar cases, both traumatic and idiopathic; and are interpreted by Rokitansky as an increase or hypertrophy of the normal neuroglia, or connective-tissue matrix.* These researches were necessarily conducted on the fresh specimen, without the help of the modern methods of research. Subsequently the organ has been examined by the method of hardening and preparing fine sections, introduced by Dr. Lockhart Clarke. Leyden,[*] who examined by this method the spinal cords from two traumatic and two idiopathic (called rheumatic) cases of tetanus, was unable to discern any morbid alteration whatever. More recently, Dr. Lockhart Clarke has observed in several cases extensive areas of disintegration in the grey substance of the cord, especially round the central canal. The degeneration sometimes amounts to liquefaction, causing distortion, accompanied by either diminution in bulk and falling in of the cord, or else swelling. He has also observed hæmorrhage. Dr. Dickinson confirms these observations, and also describes enlargement of the central canal, and production of cells in its interior; but according to Gerlach,[†] epithelial proliferation in this situation is the rule independently of disease in persons no longer young. There is also seen great fulness of the vessels, and unquestionable hyperæmia; which may probably be regarded as the consequence of increased functional activity. In other respects his description agrees with that of Dr. Lockhart Clarke.[‡]

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* This substance being naturally homogeneous, granular, and containing few formed elements, its hypertrophy is not necessarily accompanied by so much nuclear proliferation as is seen in similar increase of ordinary connective tissue, composed mainly of formed elements.

Hæmorrhage of the Spinal Cord.—An essential difference appears to prevail between the brain and spinal cord, with regard to one of the results of congestion, hæmorrhagic effusion. The frequent occurrence of apoplexy of the encephalon is familiar to all; its idiopathic occurrence in the spinal cord is extremely rare, and when brought on by external lesion, such as fracture of the vertebræ, or penetrating wounds, it is commonly associated with hæmorrhage on the surface. The cases collected by Dr. Abercrombie all appear to be instances of effusion between the meninges and the cord itself. The rarity of the occurrence may justify our extracting the following observation from the Report of the Pathological Society for 1849 (p. 28):—

"A gentleman, aged forty-four, who, with the exception of occasional attacks of gout, had previously enjoyed good health, was suddenly seized one evening with violent spasm in the stomach, and found that he had lost all sensation and power of motion in the lower half of the body. Mr. Curling found him an hour later with complete paraplegia below the third ribs, and strong priapism; no excito-motory movements were producible, and the mind was perfectly clear. The priapism subsided in about twenty-four hours; there was no extension of paralysis, except a feeling of numbness of the hands, and at last imperfect power of using them. The patient died four days after the seizure. The spine was examined seventeen hours after death. The vessels on the surface of the cord were a good deal congested. An incision was made above the front of the medulla, commencing at the part corresponding to the third cervical vertebra, and terminating at the last dorsal; two small clots of blood, amounting together to about a drachm, were found in the interior of the medulla, occupying about an inch and a-half in extent, and situated between the origins of the second and third pairs of dorsal nerves. The substance of the cord around the clots was somewhat soft; the medulla was more or less infiltrated and stained with blood from the site of the clots upwards as high as the third cervical vertebra, and downwards as low as the last dorsal."

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[*] Quoted by Rose, article on "Tetanus," in Pitha and Billroth, "Handbuch der Chirurgie," Bd. I.; 2ter abth. s. 75.

[†] Stricker's "Lehre von den Geweben," p. 686. German edition.

[‡] "On the subject of Tetanus," see Lockhart Clarke: "Med. Chir. Trans." vol. xlviii. Dickinson: "Med. Chir. Trans." vol. li.; Rokitansky: "Ueber Bindegewebswucherung im Nerven System." Wien, 1857. Similar observations have been recently published by Dr. Clifford Allbutt: "Trans. Path. Soc." vol. xxii.

No microscopic examination of the parts appears to have been made; future observation must determine whether atheromatous, or other degeneration of the arteries, or previous derangement in the nutrition of the adjoining tissues, is the causa proxima of spinal, as it so frequently is of cerebral, hæmorrhage. A case is reported by Dr. Ogle,* in which hæmorrhage took place at two points, in the dorsal region and in the medulla oblongata. The subdivisions of the vertebral arteries are described as atheromatous.

MYELITIS.

Cases of the termination of myelitis, or inflammation of the cord, in the first stage, like those of encephalitis, are scarcely ever met with; it becomes the question whether, owing to the peculiarity of the nervous structures, the first onset of inflammatory action is not at once accompanied by those changes, which in other tissues are looked upon as the secondary products of inflammation.

The product of inflammatory action most commonly discovered in the spinal cord is ramollissement, a condition which, however, like its analogue in the brain, is equally attributable to other pathological states, each of which may be recognised by the naked eye, and the aid of the microscope. The degree of softening varies from a slight diminution of consistency, as compared with surrounding parts, to a state of pulpy diffluence; the extent of cord affected differs equally. In paralysis we very frequently meet with no other trace of disease but a trifling softening in the lumbar, dorsal, or cervical regions, manifestly the result of a slow inflammatory process. The same difficulty occurs here which we met with in the brain, of distinguishing between inflammatory and non-inflammatory softening. The colour of the softened portions may be red, yellow, or white, and though granule cells or glomeruli are frequently met with, these must not be taken as a certain evidence of inflammatory action.

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* "Trans. Path. Soc.," 1853. vol. iv. p. 13.

The softening affects the grey matter, and especially that belonging to the lumbar and brachial swellings more than any other part; and a case is given by Ollivier, in which the entire grey substance of the cord was converted into a pulpy mass, leaving the white matter in a comparatively healthy state. We meet with diffused suppuration in the cord as in the brain; circumscribed abscess is also, though very rarely, found within the medullary matter. Dr. Abercrombie* gives a case of this which occurred in a woman aged fifty-six, who was affected with sudden loss of power of the limbs of the left side, followed by death in a week. The brain was sound, but in the centre of the right column of the spinal cord, in the middle of the cervical portion, there was a cavity three inches long, and two or three inches in diameter; it was full of a soft matter, like pus, which became more consistent towards the parietes of the cavity.

It appears that the softening invariably proceeds from the grey to the white matter in myelitis. It is probable that where the process is a result of exhaustion, mal-nutrition or degeneration, the reverse will be found to obtain; as the former would be favoured by the presence of a large number of blood-vessels, the latter would spread more in a part not copiously supplied with them. The tint of the grey matter is deepened, and a rose-blush pervades the white matter in the red form, while this hue is replaced by a more or less yellow tinge when the suppurative stage has set in. MM. Rilliet and Barthez have invariably found that, in children, the softening of the white matter of the cord coexisted with inflammation of the membranes, and that the extent of the former was in the exact ratio of the amount of the latter. A form of white softening occurs in the spinal cord which is analogous to the white softening met with in the brain, as a result of the effusion of serum or œdema, which is in no way connected with inflammation. Myelitis may also result in induration of the cord, but this condition is best considered by itself.

Sclerosis or Induration of the Cord.—This is found coexistent with ramollissement, or by itself. It is more frequently brought on by chronic or cachectic inflammation than softening, and it is not unfrequently complicated with hypertrophy of the affected part. When the induration is very considerable, the nerve tissue resembles, as Ollivier observes, in consistency, density, and appearance, boiled white of egg; it is a condition that Esquirol has repeatedly met with in epileptic subjects. Induration depends essentially upon the substitution for nerve tissue of newly-formed connective tissue, which appears like a hypertrophy of that previously existing; it may hence be called fibroid substitution, or more commonly, from the idea that the new tissue is inferior to, or less highly organized than the original specialized structure, fibroid degeneration. The change has also received the names of sclerosis and "grey degeneration," and is spoken of also simply as new growth of connective tissue. The process thus indicated occurs in the brain as well as the cord, but is more important and uniform in the latter. The grey colour, which is very characteristic of all forms of chronic degeneration of the spinal cord, appears to depend essentially upon the loss of the white sheath of the nervous fibres; when this is from any cause destroyed, the nervous columns appear grey instead of white. Sclerosis, or induration, appears chiefly in two forms: (1) simple induration, which affects particular nervous tracts uniformly; (2) localized induration, which appears isolated in special regions or foci, and is analogous to the local indurations observed in the brain. Simple induration is anatomically distinguished by being merely a substitution of *normal* connective tissue for nerve tissue; so that it is usually doubtful whether the atrophy of the latter or the hypertrophy of the former is the primary affection. Localized induration (which is sometimes called inflammatory) is known anatomically by the abundance of nuclei contained in the substituted connective tissue, which thus evidently owes its origin to proliferation or hyperplasia of the connective tissue, and the atrophy of nerve substance must be regarded as secondary. The adventitious sheath of the blood-vessels is especially involved in these cases, and seems to be the starting point of the growth.

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* "Diseases of the Brain," &c., 1845, p. 355.

Simple induration is especially seen in the grey degeneration of the posterior columns of the spinal cord, which is characteristic of the disease known as locomotor ataxy.

MORBID ANATOMY OF LOCOMOTOR ATAXY.

The morbid changes in this disease consist, according to the summary description of Dr. Lockhart Clarke,* chiefly in a certain grey induration and disintegration of the posterior columns of the spinal cord, of the posterior roots of the spinal nerves, of the posterior grey substance or cornua, and sometimes of the cerebral nerves. A variable number, and frequently in the latter stages of the disease, nearly all the nerve fibres of the posterior columns and posterior roots fall into a state of granular disintegration, and ultimately disappear. Usually the posterior columns retain their normal size and shape in consequence of hypertrophy of the connective tissue, which replaces the lost fibres. In this tissue lie imbedded the remaining nerve fibres, with the débris of their neighbours, in different stages of disintegration. In some places they are severed into short portions, or into rows of globular masses, formed out of their medullary sheaths or white substance, which has been stripped from their axis-cylinders. In other places they have fallen into smaller fragments and granules, which either lie aggregated in the line of the original fibres, or are scattered at irregular distances. Corpora amylacea and oil globules are also seen; the latter often collected into groups round the blood-vessels. In some cases the deeper central parts of the grey substance are more or less injured by areas of disintegration; but these latter lesions are not essential to the production of locomotor ataxy.

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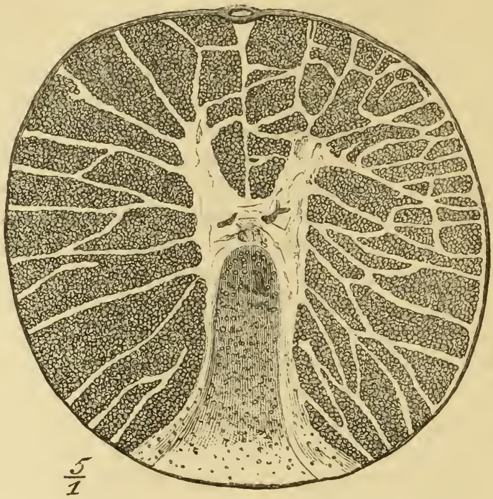
* "British Medical Journal," July 3rd, 1869.

Fig. 73.



Section of spinal cord showing degeneration of posterior columns and part of posterior cornua.
(After Leyden.)

PARTIAL OR LOCALIZED INDURATION OF THE CORD.

Sclerosis of particular regions or isolated foci occurs in the cord as in the brain. It differs from general sclerosis by the unequal knotted outline given to the cord, and further in its minute characters. It is, comparatively speaking, very rare. The hard sclerotic tissue contains a very large number of nuclei; and a similar proliferation of nuclei or small cells may be seen in the outer coats of the smaller blood vessels. The interruption of nervous currents thus caused may lead to secondary degenerations affecting considerable portions of the cord. This form of sclerosis may be regarded as analogous to the cirrhosis of the liver, granular degeneration of the kidneys, and some fibroid degenerations of other organs.*

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* Case reported by Bärwinkel, in "Archiv der Heilkunde," 1869, vol. x. p. 590 Plates xii. and xiii.

Partial sclerosis or induration with patches of grey or gelatinous degeneration, due to the new formation of connective tissue and compression of nerve substance, has also been found in different parts of the spinal cord, medulla oblongata and pons Varolii, in cases of paralysis agitans.*

Simple Degeneration of the Cord.—Degeneration of particular parts of the cord unaccompanied by new formation of connective tissue appears to occur, and to be the cause of disturbances of the functions of the cord. The precise mode in which these degenerations originate is not yet quite understood. Thus in progressive muscular atrophy or wasting palsy Dr. Lockhart Clarke† has observed at particular regions in the posterior grey matter, small areas, streaks, patches, or spots of a delicate, finely granular, transparent substance, apparently resulting from the disintegration of nerve tissue.

SECONDARY DEGENERATIONS OF THE SPINAL CORD.

When the passage of nervous impressions is from any cause completely suspended in the white columns of the cord, the substance begins to degenerate from disuse; and hence in cases of paralysis, whether central, from affections of the brain, or arising from lesion at any point of the cord itself, the whole nervous tract between the seat of disease and the peripheral termination of the nerve is liable to degenerate, and ultimately to become atrophied. In the spinal cord these changes may sometimes be traced through its whole length before there is any atrophy, and when it is perfectly healthy to the naked eye. The degenerated portions are sometimes seen on cross sections of the fresh organ as paler and more transparent, though often natural to the naked eye, than the surrounding parts; when treated with carmine, they become unusually deeply coloured. The manner in which the change may be traced and thus shown to be secondary is shown in the diagrams (Fig. 74), representing the secondary changes in a case of hemiplegia depending on tubercle in the brain.

These changes have been observed after various lesions of the brain, as embolism, tubercle, &c., but not after recent apoplexy. They also occur in consequence of special lesions of the cord itself (from tumours, &c.) below the part affected.

In the degenerated portions there was seen, beside the atrophy and fatty degeneration of the nerve fibres, what appeared to be an increase of the connective tissue, which must, of course, have been secondary to the nervous atrophy. The ultimate result is probably simple atrophy, and thus it is only in an intermediate condition that the changes just described can be traced.

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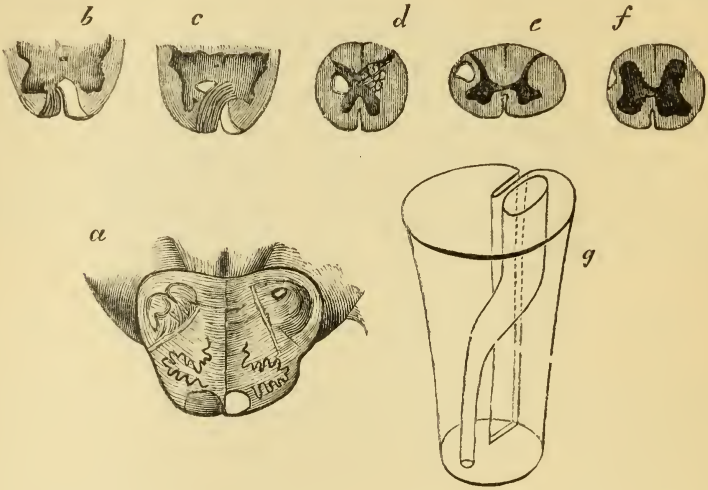
* Dr. Sanders in Reynolds's "System of Medicine," vol. ii. p. 199. Many references are given.

† Beale's "Archives of Medicine," 1861.

A similar secondary degeneration may result from some injury to the cord itself, as in a case recorded by Dr. Bastian,* where secondary degeneration of the nervous tracts was observed above and below an injury supposed to be produced by hæmorrhage into the grey matter. Below the primitive lesion, the degeneration was limited to the anterior and lateral columns; above it, to the posterior columns.

Fig. 74.



a, Pons Varolii; *b*, medulla oblongata; *c*, medulla oblongata (lower down); *d*, decussation of the pyramids; *e*, cervical portion; *f*, lumbar portion; *g*, diagram of the decussation in the pyramids.

The white areas represent sections of degenerated portion of white matter of cord.

(From Barth, "Uber secundäre degeneration des Rückenmarkes;" "Archiv der Heilkunde," 1869, vol. x. p. 433, plate ix.)

TUMOURS AND NEW GROWTHS.

Softening of the spinal cord occurs, as in the brain, as a sequel of morbid growths, such as carcinoma, or tubercle. Neither of them is, however, frequently met with, though they are oftener seen in the cord itself than its membranes. Ollivier, whose work contains the largest collection of cases of this kind on record, denies the occurrence of melanosis affecting the cord, nor have we been able to discover any other instance but the one already alluded to, in which the melanotic tumour was attached to the dura mater.

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* "Medico-Chirurgical Transactions," 1867, vol. l.

PARASITES.

Rokitansky states that he has repeatedly met with cysticercus in the cervical portion of the spinal marrow; but his experience agrees with that of Ollivier, that they do not occur in the substance of the medulla. They in most instances are situated externally to the dura mater. In this case it is manifest that they had been first developed outside the column, and had forced their way in through the intervertebral foramina; they have, however, also been found within and underneath the arachnoid.

In what Dr. Cobbold calls the most remarkable case of development of cysticerci within the human body on record, among two thousand parasites found in the body, one was in the medulla oblongata.

Hydatid cysts are very rarely found in the cord. According to Davaine, three cases have been recorded. Dr. Cobbold has met with an anonymous record of one case.

CHAPTER XV.

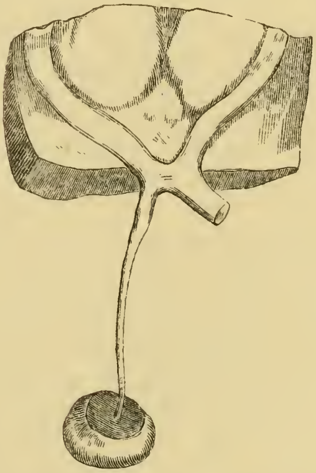
THE NERVES.

It very rarely happens that individuals die of an affection residing solely in the nerves; consequently we are left to surmise from analogy their morbid appearances in those diseases in which they are manifestly affected. At the same time, we must never forget that the nerves are not central organs, but that they are the telegraphic wires destined to convey intelligence to and from the central organs. When, therefore, we have to deal with a nervous symptom, we must first inquire whether it be due to a centric or to a peripheral cause, or, in other words, whether the nerve is propagating a morbid impression from the brain, the spinal marrow, or the sympathetic ganglia which may simulate peripheral disease, or whether it is giving evidence of local disease by producing in the brain the consciousness of that affection. In the majority of instances of irregular or painful action of the nerve, we should be as much in error in seeking for the cause of the derangement in the nerve itself, as if, when our galvanic battery does not act, owing to the trough containing no acid, we sought to remedy the defect by changing the conducting wires.

The nerves have repeatedly been made the subject of inquiry in diseases, in which either local symptoms predominate, or in which, from the known physiological action of the nerves, controlling the parts affected, the morbid phenomena could fairly be sought in an individual nerve; thus the sciatic has been subjected to examination in individuals who had been affected with sciatica, and the vagi have been explored as the hypothetical excitants of hooping-cough. Pathologists have, in neither case, succeeded in demonstrating a relation between the malady and an uniform alteration in the respective nerves. A case has long been transcribed, and has thus acquired traditional importance, by which Cotugno, the first who wrote on sciatica, is made to affirm a lesion, œdema of the nerve, as the causa proxima of that malady; but although he records a case of the kind, he himself would certainly not have approved of the interpretation which has been given to it, since he distinctly states that he attributes no importance whatever to the circumstance. With regard to hooping-cough, we find some instances recorded of the vagus having been reddened and swollen, indicating inflammatory action, but the large majority of cases in which the point has been attended to have presented no such change. Thus Dr. Albers examined the vagi, in forty-seven children who had died of hooping-cough, and found them perfectly normal in forty-three; Dr. West, who has also paid especial attention to the subject, has, only in one case out of eighteen, met with any change in the nerves; in this case they were decidedly redder than usual. We are inclined to conclude, with the latter author, that an appearance so frequently absent cannot be one of much moment, and that, like Cotugno's famous case, to which we have just referred, it may be set down to a cadaveric change.

Atrophy of Nerves.—The morbid condition most commonly seen in the nerves throughout the body is atrophy; this, however, can rarely be said to be a primary affection; it is brought on by various causes, such as the following:—(1) Pressure, acting immediately upon the nerve, and causing gradual, and even entire absorption at the point upon which the pressure acts; this we find occurring in the case of aneurism, or enlarged glands, lying in the vicinity of nerves. (2) Atrophy of the nerve results from the part to which it is supplied ceasing to perform the functions for which it receives the nerve; thus atrophy of the optic nerve may follow destruction of the eye, by mechanical injury (as in the case figured); or the nerve of an extremity wastes, when the muscles of the part are condemned to inactivity. (3) It may result from some lesion or functional inactivity of that part of the central nervous system with which the nerve is connected. In cases of atrophy and degeneration of the spinal cord, the nerves passing from the diseased portion are necessarily in an atrophic condition; thus, in an instance given by Cruveilhier,* in which the disorganization of the cord was limited to the posterior strands, extending from the lower end to the cerebellum, the

FIG. 75.



Portion of a cerebrum with the optic nerves and remains of the left eye.

The cornea is opaque, and the coats of the eye are collapsed. The left optic nerve is considerably diminished in size between the diseased eye and the optic commissure. Behind the commissure, the nerve on the right side is rather smaller than that on the left, but the thalami appear to be of equal size.

(From St. Bartholomew's Museum. Series viii. No. 5.)

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* "Anat. Pathol.," livr. xxxii. p. 19.

posterior nerves were entirely atrophied and converted into transparent threads, which contrasted strongly with the normal appearance of the anterior nerves. Special cranial nerves become atrophied when the part of the brain from which they spring is destroyed by new growth or hæmorrhage ; and anything which cuts off communication with the nerve centres has the same effect. In some of these cases, as well as in others which appear idiopathic, the atrophy appears to be preceded by an overgrowth of the connective tissue matrix of the nerve, producing a condition like the grey degeneration of the central nervous organs before described. Thus, Rokitansky[*] states, that under certain circumstances, nerves which are extremely atrophied acquire a greyish translucent appearance, especially within the skull, and that the colouring is produced by the presence of a blastema filled with numerous nuclei, which, at first gelatinous, and afterwards tough and elastic, takes the place of the nerve tubes as they disappear ; it becomes more distinctly visible, as the original neurilemma of the affected nerve diminishes. He adds, that the vessels of a nerve in this condition are often palpably dilated. (4) Atrophy may also be the consequence of inflammation or (5) of senile decay.

Hypertrophy of Nerves.—This condition is extremely rare, and its origin is quite unexplained. Perhaps the most remarkable case on record is that described by Dr. Moxon,[†] in which all the nerves of a female subject were found on an average three times the normal size. The increase depended upon an enlargement of the nerve fibres themselves, which were found to be in many cases ·08‴ or ·09‴ in diameter (while ·002 is the largest normal diameter). Virchow has endeavoured to show some connection between nervous hypertrophy, the formation of nerve-tumours, and deficient mental power. It is said that the nerves of the lower races of men are strikingly large. Hypertrophy sometimes coincides with the formation of neuromata.[‡] Enlargements of a different kind are found in nerves which are traceable to the neurilemma, or to a fibrinous deposit within the latter, as in the case of tumours of the nerves, following injury or division.

Inflammation of Nerves.—This condition is almost always brought about by injury, but may result from the extension of inflammation from neighbouring parts ; or, as is supposed, may be of rheumatic origin. The neurilemmatous sheath is the part mainly affected in inflammation ; it presents an increase of redness, of more or less intensity ; the effusion of serum induces a fulness and swelling of the nerve, and the nerve tubules themselves become separated, and, as it were, unravelled. If the production of fibroplastic matter occur, it may by compression of the fasciculi, cause their obliteration ; or, if resolution ensues, the nerve may be restored to its primitive condition, or again the part may accommodate itself to the change, and the nerve remain permanently enlarged and somewhat nodose. After partial or total division of a nerve these changes are liable to occur, and in an irritable constitution the deposit of lymph continues to act as a source of irritation, and induces intense pain. The occurrence of suppuration within the sheath necessarily gives the nerve a yellow colour, and causes the tubules to be broken up. In all inflammatory affections of the nerves, the cellular tissue surrounding them will likewise be found inflamed, It is questionable whether nerves are ever subject to any idiopathic inflammation, and when the surrounding parts are involved, these invariably attract much more attention than the nerve itself. Moreover, the statements on record, though but scanty, do not agree; thus, to return again to the vagus, which has always been the pet nerve of pathologists, probably owing to its size and superficial site, we find that Kilian has observed it to be inflamed fifteen times, in pertussis, while Breschet has only met with the occurrence twice, a relation that is the more surprising when we recollect that the sphere of observation of the former is a small German provincial town, and that of the latter, the capital of France. Autenrieth, also, states generally, that he has found the vagi inflamed, in persons who have died of spasmodic cough. The cutaneous affection called Herpes Zoster has been attributed to inflammation of a nerve branch; but there is reason to believe that the original morbid change is here in the sympathetic system.

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[*] "Lehrbuch der Pathologischen Anatomie," band. II. pp. 495, 500. (3rd edition.) "Uber Bindegewebswucherung im Nerven System," p. 21. Wien, 1857.

[†] "Guy's Hospital Reports," 1862, series III. vol. viii. p. 260, plate vi.

[‡] Virchow, vol. iii. pp. 262, 263, 265, &c.

Condition of the Nerves in Tetanus.—Two cases of inflammation of the nerve are given by Mr. Curling,[*] in his treatise on tetanus, in which healthy spots were found, between which the nerve tissue appeared inflamed. M. le Pelletier,[†] has also published several cases, in which the inflammation appeared propagated along the injured nerve, to the spinal cord, in the same disease. The most complete investigation, however, has been made by Froriep;[‡] in seven cases of tetanus, in which injury of a nerve had preceded, he has discovered a uniform lesion, resembling that indicated by Mr. Curling, and consisting in a tumefaction and reddening of isolated tracts, extending from the wound to the spinal cord; he has not found it in other cases, in which no tetanic symptoms prevailed. Rokitansky has observed in isolated spots in the track of the nerve, at very variable distances, bright red injection of the sheath of the nerve without any inflammatory products. Other careful observers as Förster[§] (in nine cases) have failed to find any such changes, so that we cannot regard them as constant.

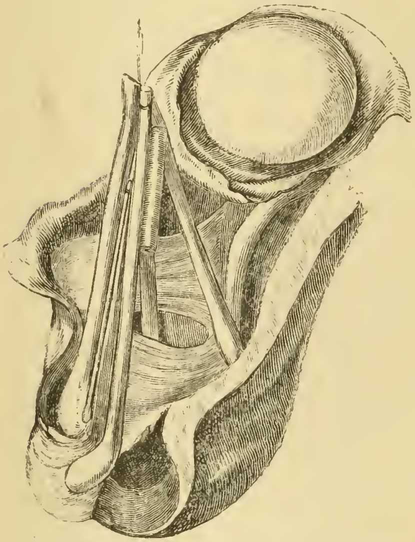
[*] "A Treatise on Tetanus," 1836, p. 72.
[†] "Revue Médicale." 1827, vol. iv. p. 183.
[‡] "Neue Notizen aus dem Gebiete der Natur und Heilkunde," 1837, vol. i. No 1.
[§] "Handbuch der Path. Anat.," vol. ii. p. 647. 2nd edition.

NEUROMA.

Of the morbid growths found in nerves, the so-called neuroma is the most common. Two kinds of nerve tumours must, however, be distinguished; the so-called true and false neuroma, the former containing true nervous tissue, while the latter is merely a tumour of some kind in connection with a nerve. The true neuromata may be composed of white medullated nerve fibres (neuroma myelinicum of Virchow) or of grey non-medullated fibres (neuroma amyelinicum). The latter are extremely difficult to distinguish from ordinary fibrous tumours, and were first separated from them by Virchow.* There is no doubt that the greater number of tumours occurring on nerves are not composed of nervous tissue.

At the extremities of the nerves in stumps after amputation of limbs, bulbous enlargements are formed, which are genuine new growths of nervous tissue. (Fig. 76.) On careful microscopical examination, the swellings are found to consist of a plexus of white nerve fibres with double outline.† Similar outgrowths are met with as the consequence of ligature of or injury to the nerve. The nerve swellings of stumps after amputation may attain the size of a bullet or plum.

Fig. 76.



A stump of the upper extremity, showing the bulbous termination of the median, internal cutaneous, musculo-spiral, and musculo-cutaneous nervous; the circumflex passes behind the teres major and its termination is not seen.

(From St. George's Museum, A. 35.)

True neuromata unconnected with division or injury of the nerve are very rare, and it must remain doubtful, from the descriptions given, whether some cases belong to this class or to that of false neuromata. They are often multiple, and sometimes occur in immense numbers on almost every nerve of the body. The spinal nerves are chiefly affected, but instances are known in connection with cranial nerves. Only the optic and olfactory nerves seem to be exempt: several cases are recorded of tumours on the auditory nerve.*

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* "Archiv," 1858, Bd. xiii. s. 263. Also "Die Krankhaften Geschwülste," Bd. iii. s. 233-305, from which great part of this section on Neuroma is derived

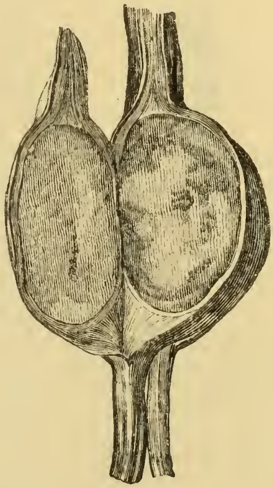
† See figure in Virchow, *op. cit.* vol. iii. p. 253, fig. 227.

Still rarer is the Neuroma amyelinicum, or tumour composed of grey non-medullated fibres; only two cases are described by Virchow himself, and we need therefore only refer to his work for further details.

False Neuromata.—The greater number of tumours of nerves belong to the classes Fibroma, Myxoma, or sometimes Sarcoma, as these all agree to some extent in their mode of growth, and it is impossible in all cases to be certain to what kind of tumour the descriptions of authors are intended to apply; we can only give a very general description, which will apply equally to many cases of true neuroma.

The idiopathic neuroma occurs without any known cause, in the shape of a round or oval tumour, varying in size from a grain of wheat to that of a pumpkin, and in number from one to several hundred. It must be classed among the non-inflammatory growths. The tumour is generally solid throughout, though occasionally it contains a cavity. It has a tendency to increase in size, and the nerve-fibres are proportionately distended and separated. It may generally be easily detached from the nerve. The structure most commonly seen is that of a dense web of fibres with scattered nuclei, coming under our definition of a fibroma. In many cases the tumour is soft, gelatinous, or semi-transparent, and would be described as a myxoma. Cystic cavities are sometimes formed, and occasionally contain a fatty emulsion, so that the tumour is of an atheromatous character. Dr. Smith † has published two cases of neuroma which are instances of the occasional extravagant production of these growths; in them almost every spinal nerve was closely studded with neuromata, which did not, however, give rise to much uneasiness; in fact it appears, as also noticed by the same author, that they are rarely productive of much pain. Another

Fig. 77.



A posterior tibial nerve, in which there is a circumscribed oval tumour, composed of a soft grumous substance.

The component fasciculi of the nerve are separated and spread out around the tumour, the peroneal nerve is adherent to the surface of the neurilemma, extended over the tumour.

(From St. Bartholomew's Museum, Series viii. No. 1.)

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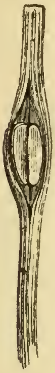
* See case by Toynbee, "Trans. Path. Soc.," 1853, vol. iv. p. 259, pl. ix. fig. 1.

† "A Treatise on the Pathology, Diagnosis, and Treatment of Neuroma," by A. W. Smith, M.D., &c. Dublin, 1849.

case of multiple neuromata from the posterior tibial nerve, in which were cystic cavities, is recorded by Messrs. Van der Byl and Snow Beck.*

Painful Subcutaneous Tumour.—This remarkable form of growth, the earliest account of which seems to have been given by Camper in 1760, though first fully described by Mr. Wood, in 1812, was at one time unhesitatingly reckoned as a neuroma. Later observers, unable to discover nerve fibres, describe it according to the structure which seemed to them predominant, as a vascular erectile tumour (Schuh), a muscular tumour, (Billroth), or a fibrous tumour (Paget). Nerve fibres can however generally be traced, and Virchow is inclined to think that some forms at least, stand in a close relation to neuroma.†

FIG. 78.



A median nerve, in which is imbedded a small tumour, over which the filaments are spread out.

(St. Bartholomew's Museum, Series viii. No. 13.)

Cancer and other malignant growths of nerves are rare, especially as a primary affection. When cancerous growth spreads into a nerve, it may infiltrate its structure without producing any very marked symptoms, simply separating, not destroying, the nerve fibres.‡ In the nerves of the senses we also meet with the primary formation of cancer. In the retina, medullary carcinoma, or some growth thus described, is not unfrequently found unassociated with cancerous growths in any other part of the system; but a primary cancer in this situation may give rise to secondary growths elsewhere. The retina is also subject to a special form of morbid growth, viz., the glioma, otherwise confined to the nervous centres,§ doubtless often described as carcinoma.

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* "Trans. Path. Soc.," vol. vi. p. 49, pl. iii., iv.

† *Op. cit.* vol. iii. p. 241.

‡ Cayley: "Trans. Path. Soc.," 1868, vol. xix. p. 53, plate iii., fig 4.

§ Virchow: "Krankh. Geschwülste," ii. p. 151. Several papers by Mr. Hulke in "Royal London Ophthalmic Hospital Reports" (vol. iv., &c.); and the textbooks of ophthalmic surgery. Also "Trans. Path. Soc.," 1867, vol. xviii. p. 230; 1870, vol. xx. p. 332.

CHAPTER XVI.

THE SYMPATHETIC SYSTEM.

A FEW cases are on record in which the ganglia of the sympathetic system were found more or less deranged and altered in structure. It is probable that a series of nervous centres, like that presented in the sympathetic, are much more frequently diseased than we have it as yet in our power to demonstrate. We can scarcely conceive that the so-called functional derangement of the heart, for instance, can continue as it does, for a series of years, and the nerves controlling its action not be or become organically altered; in the same way, long-standing derangement in the nutritive and secretive functions of the abdominal viscera may be assumed to give rise to material changes in the cœliac and semilunar ganglia, as the numerous diseases of the generative organs can scarcely exist without a similar influence being exerted upon the spermatic plexus. The anatomical disposition, as well as the physiological manifestation of the range of action of the sympathetic, justify our belief in its great and powerful agency in disease; the actual demonstration of the fact is, however, yet reserved for future inquirers. Bichat [*] repeatedly examined the nerves of the viscera in different diseases without discovering any lesions. With the exception of a single case, he has found the semilunar ganglion intact in cancers of the stomach. In a case of periodic mania he found this ganglion of the size of a small nut, with a cartilaginous centre.

Several authors have reported cases in which one or more of the ganglia of the sympathetic were found congested and inflamed in tetanus. Swan [†] has noted this condition in each of three cases in which he made a post-mortem examination of tetanic individuals. That this, however, is not a uniform lesion, and not noted because overlooked, or not attended to, is proved by the numerous cases of traumatic tetanus recently minutely examined. More or less vascularity of the sympathetic ganglia after death is so extremely common, that we must suppose it to depend upon accidental variations in the distribution of the blood supply.

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[*] "Anatomie Générale," i. 225.

[†] "On Tetanus," p. 325.

Herpes Zoster is a vesicular inflammation of the skin which, from its distribution, is clearly dependent upon a morbid condition of a cutaneous nerve, and has been sometimes regarded as a neuritis, or inflammation of the nerve trunk itself. Pain of a "neuralgic" character often accompanies the cutaneous affection, and paralysis of motor nerves more rarely. Actual changes in the nerve twigs are somewhat dubious (though described by Haight), but more decided changes have been traced in the sympathetic ganglia connected with the nerves. The nerves most commonly affected are the branches of the fifth cranial, the intercostals, and in the leg, the anterior crural or other branches of the lumbar plexus. The ganglia corresponding to these are the Casserian ganglion for the fifth, and the ganglia connected with the posterior roots of the spinal nerves. The latter were long ago described by Bärensprung as congested in cases of intercostal herpes ; and lately V. Wyss has, with the aid of modern methods of research, examined the Casserian ganglion in a case of herpes of the fifth cranial nerve, and found it acutely inflamed. We must, therefore, suppose that the disease is one beginning in the grey matter of the ganglia, and that some morbid change peripherally transmitted, causes the cutaneous eruption.

It appears that the sympathetic system may also, though very rarely, be the seat of neuroma. Dr. Smith gives an instance of it occurring in the cervical ganglia, and figures it ; it is, probably, the same case which is described by Cruveilhier,* as a case of fibrous transformation, and enormous development of the cervical ganglia, and the nerves of communication passing between them. One of the tumours was two and a-half inches long by one in breadth. Both authors state that their subject was accidentally discovered in the dissecting room, in Paris, and that no history of the case was obtainable.

Dr. Smith, in alluding to this remarkable degeneration of the cervical ganglia, states that, according to Schiffner and Bischoff, this condition of the sympathetic frequently coincides with idiotcy and cretinism ; it is a point which requires further confirmation. Pinel (quoted by Virchow) is said to have found the superior cervical ganglia of the sympathetic enlarged to three times the normal size in nine insane persons.

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* "Anat. Pathol.," Livr. pl. iii.

THE PATHOLOGICAL ANATOMY OF THE ORGANS OF CIRCULATION.

CHAPTER XVII.

THE MORBID ANATOMY OF THE PERICARDIUM.

THE frequency of morbid alterations in the pericardium increases with age, a proclivity which is not marked in the same degree with regard to the internal lining of the heart, which is much more prone in childhood to take on diseased action than we should be inclined to assume à priori. Congenital affections of the pericardium are very rare, and though cases of its entire absence are recorded by observers like Baillie and Breschet, the majority of instances that have been classed under this head have been shown to be only apparent anomalies, owing to intimate adhesion between the two surfaces of the sac giving rise to the semblance of the defect. A case of undoubted absence of the pericardium, where the heart lay in the same serous sac with the left lung, was observed by Dr. Baly in a man aged thirty-two.*

Another congenital peculiarity rarely met with is the non-attachment of the pericardium to the diaphragm, so that the former constitutes a distinct pendulous bag. Congenital openings into the pleura have also been observed.†

Fibrous thickenings of the pericardium may occur so early in life as to seem congenital; we allude to the so-called milk spots, which are yet considered by various authors as results of inflammation exclusively. This is the view of Mr. Paget, while Dr. Hodgkin is inclined to look upon them as the product of attrition only, as they are almost universally found on the anterior surface of the heart, at the point most in contact with the anterior walls of the thorax. The discrepancy is probably reconciled by the observation that there are two kinds of white spots, as was well laid down by M. Bizot;‡ the one, probably, owing to a previous attack of pericarditis, the other to simple thickening of the pericardium from friction. They are cream-coloured opacities of the visceral layer, varying in size from that of a sixpence to that of a five-shilling piece, and more, which may at times be detached from the subjacent serous membrane, to which they are then connected by fibrous adhesions. In other cases they are mere thickenings of the pericardium itself, or rather of the subserous cellular tissue, and cannot in that case be detached from it. The former, to use the accurate description of M. Bizot, present at their commencement the form of small transparent elevations, aggregated together with circumscribed edges, and but slightly adherent to the serous membrane. They soon lose their transparency, becoming white and opaque, but still capable of being removed without injury to the subjacent serous membrane. They occupy every portion of the heart, but lie chiefly in the direction of the blood-vessels. The second variety have no circumscribed margin, they are peculiarly white, and their greatest thickness is in the centre from where they are bevelled off in all directions; these are essentially identical with the pericardium itself, and constitute a true hypertrophy of the membrane. We are ourselves of opinion that they are to be viewed as resulting from two causes; and that while they are in many instances the results of previous inflammation, they may also represent a simple hypertrophic condition resembling the horny thickenings of the cutis. The great frequency of the occurrence of milk spots* in Bright's disease of the kidney has been pointed out by some observers, and is obviously connected with the frequent hypertrophy and enlargement of the heart in that disease. Milk spots of this kind are always found on those parts of the heart which are exposed to friction. Thus, when the heart is normal in size, the most frequent situation is near the apex, on the area which touches the chest wall. Another situation is the front aspect of the right ventricle, which, in dilatation of the heart, frequently strikes against the walls; and other portions of the ventricular or auricular surface are, for similar reasons, sometimes thus affected.

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* "Report of Pathol. Soc.," 1851, p. 60.

† Douglas Powell: "Trans. Path. Soc.," 1869, vol. xx. p. 99.

‡ "Mémoires de la Société d'Observation," vol. i. p. 347.

PERICARDITIS.

The first effect of inflammation in the pericardium is to produce a reddening and pulpy thickening of the membrane, by the congestion of the blood-vessels and interstitial effusion of serosity. A beautiful reticulation of minute vessels is visible to the naked eye, and still better under a low power of the microscope; both on the internal and external surfaces of the pericardium "exudation" on the free surfaces then follows, which, according to the constitution of the individual, is of a more or less plastic character. In the most active forms, the matter produced is a semi-fluid, organizable material of a yellowish hue, forming a reticulated or villous appearance, which must be attributed to the movements of the heart. Laennec has very correctly compared the appearance thus presented to that we see on quickly separating two slabs of marble, between which a layer of butter was interposed. This plastic material gradually becomes organized, and we find minute red vessels projecting into it; and as this process proceeds the two surfaces become intimately adherent to one another; the lymph loses its fluid constituents; it is converted into firm bands, connecting more or less loosely the visceral and parietal pericardium, which, according to their density and tenacity, indicate the period of their formation. If adhesion does not result, absorption may remove these appearances, and nothing but a general opacity or thickening of the pericardium remains; or again, the active condition may be arrested after the formation of villi, and without the supervention of adhesion they may continue in a passive state, and present the appearance termed the hairy heart, the cor villosum. It is customary to quote as an instance of this a classical name; the great enemy of Sparta, Aristomenes, was captured and killed on his third entry into Lacedæmon, and his heart is stated by Pausanias to have been found covered with hair. In other cases the effusion resulting from pericarditis will be of a

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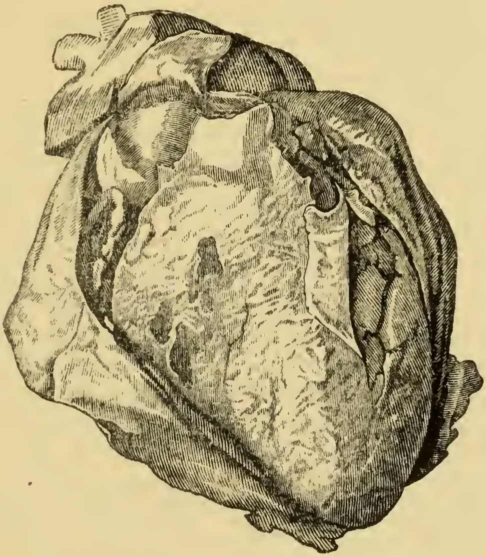
* See Dr. Taylor's paper on the Causes of Pericarditis, in "Medico-Chir. Trans.," vol. xxviii. p. 468.

Fig. 79.



A heart covered with inflammatory lymph, investing both the parietal and visceral layer of the pericardium, which has been cut open and reverted.

An incision has been made through the false membrane over the left ventricle, to turn it back and show the subjacent muscular tissue. The lymph fringes the right auricle and coats the root of the aorta.

more serous character; and we then find the pericardium more or less distended with a straw-coloured fluid, in which flakes of lymph are discovered, while traces of lymphatic exudation are seen attached to the membrane with thin free ends waving in the fluid. We have seen the pericardium mount up from this cause to the second rib, and the quantity of serum varies from half an ounce to two quarts. In this case, as in the former, absorption may take place, leaving but comparatively slight traces of the previous disease, and the pericardium itself appears to adjust itself to the reduced quantity of its contents.

A third form of pericardial inflammation is the purulent. Fluid containing larger or smaller amounts of pus is found distending the sac. This form is, of course, connected by insensible gradations with the others, inflammatory exudations differing from one another, as we have before mentioned, chiefly in the larger or smaller proportion of emigrated blood corpuscles or pus cells. The purulent form is chiefly found in cases which have lasted for some time, and tend to assume a chronic form, though it is thought by some that it may be suppurative from the first. This form of pericarditis occurs especially in weakly constitutions, and those that are disposed to so-called scrofulous affections.

Minute Anatomy of Pericarditis.—The formed elements in the inflammatory lymph of pericarditis consist in the first place of corpuscles resembling leucocytes, many of which we may conclude have emigrated through the walls of the blood vessels; and in the second place of more complex elements which have been especially studied by MM. Cornil and Ranvier. They are cells containing large oval nuclei, with brilliant nucleoli, resembling the nuclei once thought characteristic of cancer cells. The cells themselves are sometimes flattened like those of serous epithelium, sometimes showing prolongations; some possessing a large number of nuclei, resemble the myeloid cells or "myéloplaxes" of bone, which have also been seen in various morbid conditions of the serous membranes, and especially in tubercle. These cells are found in sections to be arranged in layers parallel to the surface, and alternate with layers of fibrine arranged in an irregular fashion. They conclude that probably all these cells proceed from cells of the serous epithelium, which swell, proliferate, and ultimately become detached.*

Similar changes, viz., swelling, division of the nuclei, and proliferation of the epithelial cells, have been seen by Dr. Chapman† (following the method of Cohnheim) on the pericardium of the living frog. He also observed amæboid movements of the cells themselves, and changes resulting in the production of cells of the most various kinds, including pus cells. Proliferation was also observed in the connective-tissue cells. So that, as in other inflammations, we may conclude that the fixed elements of the tissue contribute to the formation of new elements, though some of these may be leucocytes which have escaped from the vessels.

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* Cornil and Ranvier: "Histologie Pathologique," p. 459.

† "American Journal of the Medical Sciences," October, 1872.

Hydropericardium.—The serous effusion just spoken of must not be confounded with the dropsical accumulation of fluid, to which we should restrict the term hydropericardium, and which is a frequent accompaniment of general dropsy. In many cases of wasting disease we find a small quantity—perhaps one or two ounces—of serum in the pericardium, which appears to be eliminated shortly before death. It is not associated with symptoms of inflammatory action; and the fluid itself is a clear, amber-coloured serum. The amount appears to increase by post-mortem transudation. In long-continued dropsy of the pericardium the heart is generally found contracted, and the muscular tissue anæmic and of light-brown hue. Occasionally an atrophic condition of the muscular fibre results, which is characterized under the microscope by an absence of the striation seen in healthy muscle.

Results of Pericarditis.—The phlogistic process in the pericardium generally involves the entire surface of the membrane in acute cases; the chronic form, except as a sequel of the former, has a tendency to limitation, and its residuary effects are seen in the form of circumscribed white patches, either on the visceral or parietal portion, or of partial adhesions or isolated bands of false membrane.

The false membranes remaining after an attack of pericardial inflammation, may, unless absorbed, become the seat of changes similar to those which we find them undergoing in other structures throughout the body. They present a metamorphosis into fibrous, cartilaginoid, and osseous tissue. The deposit of the latter occurs in smaller or larger patches; they may be numerous and distinct from one another, or they may unite to form, as in a preparation contained in Guy's Hospital (No. 1,448), a complete ring encircling the base of the heart.

Calcification sometimes goes so far as to affect the whole of the parietal layer, producing the effect of a bony capsule enclosing the heart.* True bone is said to be sometimes formed.

Causes of Pericardial Inflammation.—Pericarditis is not often an idiopathic disease. Dr. Latham, who was the first to notice its frequent complication with the rheumatic diathesis, has rarely met with it except in this connection. Andral gives six cases of pericarditis not connected with rheumatism, of which three were uncomplicated with any other morbid affection; while Corvisart only met with five independent of rheumatism, which were all, excepting one, complicated with disease of other parts. The rheumatic complication is one found at all periods of life. Messrs. Rilliet and Barthez and Dr. West look upon it as essential in young children; and all writers on the subject concur with regard to its frequency in adults, though the statistical results arrived at are not perfectly uniform. A further powerful predisposing cause is to be found in renal disease, and more especially in that form known as Bright's disease of the kidney. Dr. Taylor gives the following results of the analysis of the causes of thirty-seven cases of acute pericarditis:—

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* "Trans. Path. Soc.," 1869, vol. xx. p. 101.

There was rheumatism in	20	cases.
Bright's disease in	10	,,
Bright's disease doubtful, or other form of renal disease in	5	,,
Cyanosis	1	,,
Extension of inflammation from adjoining tissues in	1	,,
	37	

It follows that in examining the dead subject, we should, in all cases of pericardial disease, be particularly careful not to omit looking to the condition of the kidneys, even if the symptoms during life were not such as to draw the physician's attention to these organs. Considering the degenerative character of Bright's disease, and its chronic course, we are justified in regarding it as a powerfully predisposing cause to inflammation of serous membranes, and particularly of the pericardium. Pericardial inflammation is also met with in cases of pyæmia, and is sometimes consequent upon other inflammations such as necrosis of bone, abscess, and the like, in cases where no other manifestations of pyæmia are observed. It also occurs, though rarely, among the sequelæ of scarlatina in children.

HÆMORRHAGE.

In the exudation resulting from acute inflammation we occasionally meet with a small quantity of blood. Hæmorrhage, independently of this cause, of mechanical injury, or of rupture of the muscular tissue of the heart, is not so often met with in this locality as it is in the sac of the arachnoid. When it occurs it is due to the rupture of a branch of one of the coronary arteries, or of an aneurism developed upon it. In a case reported by Dr. Ogle,* the extravasated blood had formed a cyst, about three inches long, chiefly in the visceral layer but to which the parietal layer had become adherent. In all the cases we have observed there was atheromatous disease of the coronary arteries. Minute aneurisms like those of the cerebral arteries have also been observed by Liouville. Aortic aneurisms sometimes open into the pericardium.

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* "Trans. Path Soc.," 1858, vol. ix. p. 165.

TUBERCLE.

The pericardium does not resemble the other serous membranes, in its relation to tubercle; for while the meninges of the brain and the peritoneum are constantly found to be the seat of tubercular deposit, this serous sac is remarkably free from it. Louis * has only found evidence of pericarditis three times in phthisis; and he details one case in which some semi-transparent grey granulations were found under the serous lamina of the pericardium, to which he attributes the pericarditis under which the patient was labouring. Dr. Hope states that tubercles are sometimes developed in the false membranes of pericarditis; but neither does he himself give any positive evidence to that effect, nor have we succeeded in finding proofs of it elsewhere.

In cases of general acute tuberculosis, especially in children, miliary tubercles are sometimes seen on the visceral pericardium.

CARCINOMA.

Carcinoma affects the pericardium more frequently than tuberculous disease; it occurs only in connection with a general cancerous cachexia, and a formation of similar growths in other organs; the only variety met with is medullary carcinoma. According to Rokitansky the cancerous mass spreads itself in the form of an infiltration of the fibrous layer of the pericardium over a large portion of its surface, and presses upon and into the tissue itself, where it becomes developed into roundish or flattened or teat-like nodules. This description partly agrees with a case reported by Dr. Peacock, in which the organ was completely surrounded by a cancerous mass, which did not, however, affect the muscular substance. Malignant growth not unfrequently extends from the mediastinal glands into the pericardial cavity; but many such cases would at the present day be called sarcoma or lympho-sarcoma, rather than cancer.† Several are noticed by Dr. Peacock, in the paper just referred to.‡ Förster observed one case of primitive cancer of the pericardium. A malignant growth of the pericardium, reported by Dr. Moxon, was found to be, anatomically, a sarcoma.§ A fibroid tumour of the pericardium is noticed by Dr. Chambers.|| In some of these cases the morbid growth may even infiltrate the walls of the heart, from the pericardial surface, and project into the cavities.

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* "Mémoire sur la Péricardite," &c., 1826.

† Dr. Church in "Trans. Path. Soc.," 1869, vol. xx. p. 102.

‡ "Trans. Path. Soc.," 1865, vol. xvi. pp. 99-120, and plate v.

§ "Trans. Path. Soc.," 1867, vol. xviii. p. 38; Report by Hulke and Cayley, p. 41, figs. 3 and 4; see "Trans. Path. Soc.," vol. ix. p. 327, for another case.

|| "Medico-Chirurgical Review," October, 1853.

FATTY DEPOSIT.

It is not uncommon to meet with an excessive deposit of fat upon and within the pericardium; it occurs in conjunction with general obesity, as well as in cases where there is little subcutaneous fat; nor is it necessarily associated with true fatty degeneration of the muscular tissue of the heart, though we may at the same time find fat insinuating itself into the heart so as to separate the muscular fasciculi from one another. It will be observed that accumulation upon the heart is largest in the horizontal sulcus, and that its distribution appears to bear a relation to the arrangement of the blood-vessels.

PNEUMO-PERICARDIUM.

A condition of the heart rarely found until after death, and termed by Laennec pneumo-pericardium, consists in an effusion of air into the sac. Laennec states that he was able to diagnose its presence during life from the unusually clear sound yielded by percussion in the region of the heart, and by a sound of fluctuation accompanying the movements of the heart and of respiration. In the majority of cases it is due to post-mortem decomposition of the pericardial fluid, and when this is not the case the air has found its way into the pericardium from some adjacent cavity. Cases of supposed spontaneous generation of gas in the sac during life rest on very insufficient evidence, though it is possible this may occur from the decomposition of foul pus. During the present year (1852) a case of perforation of the œsophagus, which had formed adhesions to the pericardium, occurred in St. Mary's Hospital. The patient was a young woman under the care of Dr. Chambers, in whom the admission of air into the pericardium occurred shortly before death through the perforation; the pericardium was found much distended from this cause, when the post-mortem examination was made; and it was owing to this circumstance that the fibrinous layer, which had been deposited between the surfaces of the pericardium, had not given rise to any friction sound during life.* Ulcers of the stomach have produced the same condition.

FREE BODIES IN THE PERICARDIUM.

To complete the subject of the morbid contents of the pericardium, we have yet to advert to the presence of free bodies, which Rokitansky has met with in a case of pericarditis. He describes them as fibrinous, soft, yellow concretions, of the size of beans or almonds, and similar to the latter in shape; which, he adds, would no doubt have eventually been converted into elastic, tough bodies of fibroid tissue. None of the authors whom we have been able to consult record any similar case; we may therefore assume that the actual occurrence of free bodies is a circumstance of extreme rarity, and the above seems rather to be due to an accidental agglomeration of fibrinous flakes than to any other new production of tissue. In other cases pedunculated fibrous growths, sometimes even calcified, appear to have become detached, by giving way of their peduncle, and thus come to lie free in the cavity of the pericardium.

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* See "Report of the Path. Soc.," for 1852-3.

CHAPTER XVIII.

THE MORBID ANATOMY OF THE HEART.

CARDITIS OR MYOCARDITIS.

GENUINE carditis, ulceration, and abscess of the heart are conditions of which but few cases are recorded. Of the former Dr. Latham* details a remarkable instance. It occurred in a boy, aged twelve years, who presented all the symptoms of acute cerebral disease, without any indications of the disorganization found after death. No vestige of morbid action was discovered in the brain, but the heart was the seat of the most intense inflammation, pervading both the pericardium and muscular structure. There was the ordinary evidence of recent pericarditis, and when the heart was itself divided, the muscular fibres were dark-coloured, almost to blackness, loaded with blood, soft, and loose of texture, easily separated, and easily torn by the fingers, and at the cut edges of both ventricles small quantities of dark-coloured pus were seen among the muscular fibres. The internal lining was of a deep red colour, without any effusion of lymph. A case which resembles the one just detailed, and which occurred in the practice of Mr. Salter, of Poole, is detailed in the twenty-second volume of the "Medico-Chirurgical Transactions." In this case the principal morbid change was in the substance of the left ventricle, which was of lightish yellow hue, still preserving the fibrous character of muscle. From all the cut surfaces purulent matter could be scraped; in some parts absorption had taken place, leaving small cavities in the muscular substance, varying from the size of a pin's head to that of a small pea; these were all filled with pus. A third case of the same kind is related by Mr. Stanley in the seventh volume of the "Medico-Chirurgical Transactions," p. 323, which occurred in a boy aged twelve. According to Förster, the left ventricle is almost exclusively the seat of this disease, its occurrence in other parts being exceptional. The muscular fibres are found affected even in an early stage of the disease, becoming pale, granular, and losing their transverse situation. The amount of exudation or inflammatory material produced is not great, and is chiefly serous; but sometimes, as in the instances just mentioned, true suppuration takes place. The pus cells are in this case produced in the interstitial connective tissue between the muscular fibres, and there is no reason to think that the muscular elements themselves participate in their production. The loss of substance due to inflammation may, if it occur immediately under the surface of the heart wall, tend to a kind of ulceration; and in this case there is always simultaneous inflammation of the endocardium. If the same process affect the septum ventriculorum, it may lead to actual perforation. In a case of general hypertrophy of the heart, accompanied by enormous dilatation of the mitral orifice, and diseased aortic valves, which was exhibited at the Pathological Society in 1847, by Dr. J. R. Bennett, an opening of the size of a quill was found in the ventricular septum; this was surrounded by ulceration, warty roughness, and thickening, and there were distinct traces of inflammation round the opening. Perforation may also result from the opening of an abscess externally, which produces pericarditis and rupture of the heart. Incomplete softening and destruction of the walls of the heart lead to the condition known as *aneurism of the heart;* that is, to partial dilatation and attenuation of the wall. It is, however, possible for the pus to become incapsulated, when it either undergoes caseous or calcareous degeneration, or else is absorbed altogether.

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* "Lectures on Clinical Medicine," &c. 1845, Lect. xxv.

In general pyæmia, purulent collections are found in the heart, though not frequently; they do not differ from those which occur in other organs.

Other causes sometimes give rise to suppuration of the heart. In a case recorded by Dr. Pye Smith,* this condition seems to have resulted from the breaking up of syphilitic growths.†

NON-SUPPURATIVE MYOCARDITIS.

There is a form of inflammation of the heart substance without suppuration, which, in a chronic form, leads to the changes described in the next paragraph. It is uncertain whether it is ever strictly acute, but when recent, produces the following appearances. The muscular substance is soft, and mottled with yellow patches, which consist not of inflammatory products, but of muscular tissue in a state of colloid or vitrous degeneration, the striation being indistinct, and the fibres atrophied. Around and among the wasted muscular bundles is much vascular nucleated tissue. This process is anatomically comparable to cirrhosis of the liver, and bears the same relation to chronic myocarditis that rapid cirrhosis does to chronic. It never occurs without accompanying endocardial (and usually) pericardial inflammation, and though doubtless the first stage of chronic myocarditis, does not very often come under observation.

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* "Trans. Path. Soc.," vol. xxi. p. 94.

† For other cases of inflammation or abscess of the heart, see Murchison: "Trans. Path. Soc.," 1868, vol. xix. p. 193; Moxon: Ibid. 1869, vol. xx. p. 113; Holmes: Ibid. 1858, vol. ix. p. 164.

CHRONIC MYOCARDITIS.

A much commoner result of myocarditis than suppuration is the formation of fibroid masses. This is very common in the hearts of rheumatic subjects who have suffered from pericarditis or endocarditis, and depends upon the production of connective tissue, concurrently with (whether as consequence or cause) the loss of muscular substance from inflammation. It is usually partial or in separate spots or streaks, but is sometimes pretty generally diffused. The parts affected are pale, firm, glistening, and sometimes of almost cartilaginous hardness. The microscope shows that they consist of dense fibrous tissue, usually with a superabundance of nuclei, but sometimes giving evidence of degenerative changes. While this is doubtless the explanation of the fibrous patches usually met with in diseased hearts, there are more pronounced new formations and more general changes of the same kind, which will be considered elsewhere.

Myocarditis probably never occurs without inflammation of the external or internal membrane of the heart, except as a consequence of pyæmia. Dr. Wilks attributes to incipient inflammation of the muscular substance certain cases of sudden death in rheumatism. It should be mentioned that the columnæ carneæ are very liable to be affected with fibroid transformation, and may become altogether converted into fibrous cords; this condition usually accompanies valvular disease.

Hæmorrhage into the substance of the heart is occasionally met with in small spots, in connection with pericarditis; but in cases of genuine cardiac apoplexy, as Cruveilhier terms effusion of blood into the substance of the heart, which must be considered in the same category as spontaneous rupture of the organ, we almost invariably find fatty degeneration at and near the point, which has destroyed the uniform consistency of the organ.

DEGENERATIONS OF THE SUBSTANCE OF THE HEART.

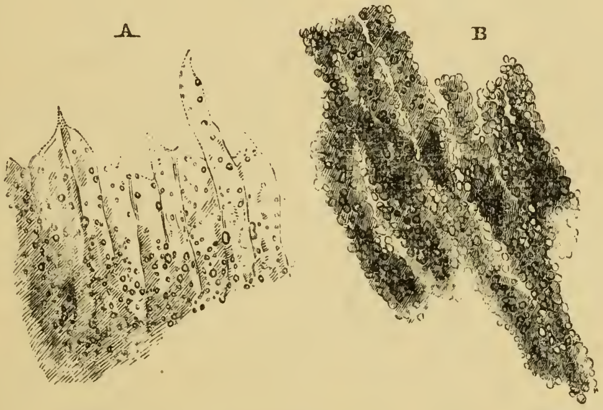
Under this head are included fatty degeneration, fibroid degeneration or transformation, and a much rarer condition, granular degeneration or "brown atrophy." Waxy or amyloid degeneration is also met with, though very rarely.

Fatty degeneration is a condition of the muscular fibres themselves, which can only be positively recognised by the help of the microscope.

The degeneration may be traced through various stages. It commences as an atrophic condition, in which the fibre loses its sharp edges, and the striæ, so well seen in the healthy heart, disappear. One or more oil-globules successively appear, until the whole fibre is occupied by them; its outline is broken, and in the highest development the fibres appear almost fused together into a confused more or less opaque mass, in which nothing of the original tissue can be traced. This molecular deposit of oil in the fibre is often accompanied by a generally adipose condition of the organ, and a layer of fat on the surface; but not necessarily so, and we must be careful not to infer that the heart is in a state of fatty degeneration because it is surrounded by adipose tissue. The fat may insinuate itself between the muscular fasciculi and fibres, and yet no change in the latter take place. The large circular fat cell, with its sharp boundary of $\frac{1}{500}$ inch in diameter, when seen under the microscope, occupies the former position, and cannot be mistaken for the minute oil-globule, which varies from a microscopic point to $\frac{1}{5000}$ inch, and is confined to the interior of the sarcolemma.

The heart affected with fatty degeneration has lost the firm muscular appearance which characterizes it in health, and presents a pale, yellowish, buff colour, either throughout or limited to individual parts. When cut into, a greasy stain is often left on the knife, though the absence of this circumstance must not be looked

Fig. 80.



Specimens of fatty degeneration of the heart.

A, Heart fibres taken from the columnæ carneæ of the mitral valves of a young woman aged thirty; the fatty degeneration was scarcely observable in the ventricle, where the fibres still retained their striæ. B, An extreme case of fatty degeneration, showing an entire conversion of the muscular fibre into oil molecules, still retaining a linear arrangement; it is taken from the right ventricle of an old gentleman, who had Bright's disease of the kidney and pulmonary phthisis, and was affected with fits during the last two years of his life.

upon as an indication that the heart is not degenerated. The left ventricle and the columnæ carneæ are most liable to be thus diseased, next in order the right ventricle and right auricle, while the left auricle is least frequently involved. Dr. Quain,* to whom we are indebted for a clear *résumé* of the whole subject of fatty degeneration of the heart, has found that in twenty-two cases in which the seat of the disease was expressed, the two ventricles were affected in ten, the left alone in eight, and the right alone in four. Rokitansky describes three forms of fatty degeneration of the heart; the first two are varieties of fat-deposit upon and within the heart, external to the muscular fibre to which we have alluded above; and to which, with Dr. Quain, we would apply the term fatty growth, while the term fatty degeneration should be confined to that change in the muscular fibre which we have just described, and which constitutes Rokitansky's third form. The deposit and the degeneration may coincide, but there is no definite relation between the occurrence of the two.

The frequency with which fatty degeneration of the heart occurs among the patients that present themselves in a London hospital may be inferred from the circumstance that Dr. Ogle has met with it in 100 out of 143 post-mortems in which he had noted the microscopic appearances of the organ, a circumstance sufficient to rivet the attention of nosologists upon the heart, in order to determine with more accuracy than we at present can bring to bear, the incipient morbid conditions to which this state is due. It is essentially a disease of middle and advanced life; and is, we may say, invariably associated with a fatty condition of other organs, more especially of the liver, the spleen, and the arterial system. Dr. Quain has laid much stress upon affections of the coronary arteries accompanying fatty degeneration of the heart substance. Rigidity and constriction of these arteries, dependent upon atheroma, seriously diminish the supply of blood to the organ; and it is often found that the most extensively diseased branch of the artery supplies that part which has undergone most complete degeneration. This condition of the arteries may, however, be due to the same cause as the degeneration of the muscular substance rather than a cause of it, and be evidence of a degenerative process affecting the whole body; it is thus most commonly met with in old age. Another sign of this constitutional state is the *arcus senilis*, the frequent coincidence of which with fatty degeneration of the heart was first pointed out by Mr. Canton. This coincidence is, however, not sufficiently constant to enable us to form a positive conclusion as to the condition of the heart from the appearance of the cornea. Fatty degeneration is also sometimes connected with a special diathesis, as scrofula or syphilis, and may be due to some special poison, as phosphorus.

Syncope and angina pectoris during life are among the effects of fatty degeneration of the heart and diseased coronary arteries; apoplectic effusion into the substance of the organ, rupture, dilatation, and aneurism of the heart, are found in constant connection with this affection. It is thus one of the most important causes of sudden death.*

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* "Medico-Chir. Trans.," vol. xxxiii. p. 121.

Fibroid degeneration, or transformation of the heart substance, usually occurs in cases of hypertrophy. Dr. Wilks has pointed out that it is far more common in the right ventricle than in the left, when thus affected; and that in most cases of hypertrophy of the left ventricle there is no change in the consistency of the walls. While this is doubtless true, we believe it to depend upon the fact that so many cases of left-sided hypertrophy are due to some cause outside the heart itself. When this is due to valvular disease, or accompanies endocarditis, or pericarditis, the left ventricle also may be found much altered in consistency. In this form of degeneration the muscular substance is pale, as in fatty degeneration; but hard and rigid, so that the cavities retain after death the form due to distension. It is also torn with difficulty, and has been described as leathery. Minute examination shows these changes to depend on replacement of the muscular fibres by newly-formed fibrous tissue, so that the condition differs chiefly in its universality from that previously described as the result of myocarditis. A heart thus affected is evidently as little capable of performing its functions as one in which there is fatty degeneration; and hence this condition also appears to be a cause of sudden death. It is commonly associated with enlargement of the heart; and the question will sometimes arise whether the fibroid growth causes the enlargement, producing a false hypertrophy; or whether it is a further result of the same pathological process as that which has produced the hypertrophy itself.

Granular degeneration, or brown atrophy of the heart substance, is a rare condition; but one of which unquestionable instances sometimes occur. It consists in the deposition of brown particles between the fibrillæ of the muscular substance. This seems to depend originally upon a sort of fatty degeneration, fat being deposited, and then metamorphosed into these brown granules. (Virchow).†

Waxy, or *amyloid degeneration* of the heart substance, is very rarely met with, and only accompanying the same condition in other organs. We have seen but one case. The name waxy, or bacony degeneration, has also been given to the condition which we have described as fibroid.

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* Numerous cases bearing on this subject are noticed in "Trans. Path. Soc.," 1853, vol. iv.

† MS. notes of lectures.

APOPLEXY AND RUPTURE OF THE HEART.

Without a knowledge of the changes which have taken place in the tissue, and precede the occurrence of these accidents, it is impossible to offer any rationale for them; but now that we are acquainted with the fact that the muscular fibre is degenerated at certain parts of, or throughout the organ, it is easy to understand that, under given circumstances, requiring an unusual effort in the heart, the weakest point will yield, and give rise to effusion of blood. There is no essential difference between those cases in which the hæmorrhage seems confined to the muscular tissue and those in which, owing to a laceration of the pericardium and endocardium, a passage is established by which the blood flows into the serous sac. In the latter case we find the pericardium, on opening the body, distended with fluid blood, or, if the individual has survived some time after the accident, the blood is partly coagulated. The rent varies from an inch in length to a minute orifice; it frequently runs into the septum, and occasionally we find an accompanying rupture of the columnæ carneæ. The left ventricle is by far the most frequent seat of these disruptions; we find that six or seven cases of spontaneous rupture of the heart, detailed in the Reports of the Pathological Society of London, occurred in the left ventricle, and only one in the right. An analysis of these cases also shows that the prevailing impression that the anterior surface is more liable than the posterior to become lacerated, is erroneous; five having occurred on the posterior, and two on the anterior walls of the heart. In all there was fatty degeneration, most marked at the seat of injury; the coronary arteries were found in an atheromatous or ossified condition in the five cases in which they were examined; the average age of the sufferers was 69¼ years. A rather different result is obtained by an analysis of cases of rupture of the heart, following mechanical injury, without penetrating wounds; here there is no suspicion of fatty degeneration, and a different explanation must be sought for, to account for the seat of the rupture, which appears to vary as much as the injury itself. We find, that of five cases of this description, in all of which there was no penetration of the heart's substance from without, one occurred on the posterior surface of the left ventricle, one on the posterior surface of the left auricle, two on the anterior surface of the left auricle, and one on the anterior surface of the right ventricle. Here, the left auricle was three times affected, and each of the ventricles once.

Rupture of the heart is generally immediately fatal; instances are, however, recorded in which the patient recovered from the first shock, and survived for several hours; in these cases Nature is found to have made an effort at repair, in the shape of a film of lymph, exuded between the torn surfaces.

Gangrene of the heart is a subject alluded to by pathologists, but it does not appear that any authentic cases of its occurrence are recorded. Dr. Copland looks upon it as manifestly a post-mortem alteration, accelerated by a depraved habit of body. We may, therefore, at once pass to the consideration of two conditions which are very frequent, and which are nearly allied to one another, hypertrophy and dilatation of the heart.

CHAPTER XIX.

HYPERTROPHY OF THE HEART.

In determining the existence of hypertrophy of the heart we must attend to two preliminary points: first, we must ascertain whether there is an absolute increase of the total bulk, as compared with hearts of healthy individuals of the same age and conformation; and, secondly, whether the relative size of the walls of the different cavities has altered. Next, it will be well to inquire into the relation existing between the walls of the cavity and its capacity; and it is also necessary to remove the contents, fluid or consistent, that may distend the cavities, before we form our estimate. Laennec suggested that the doubled fist of the individual might be taken as a rough measure of the size of the heart; as he found that, in health, the two corresponded in their dimensions; there is no objection to retaining this indication, to assist our judgment, when more accurate determinations are not at our command.

NORMAL WEIGHT OF THE HEART.

The results of Dr. Peacock,* arrived at from the examination of 198 hearts regarded as healthy, and 150 in some way diseased, are as follows:—

1. The average weight of the healthy heart in persons from 20 to 55 years of age, is in males 9 oz. 8 dr., and in females 8 oz. 13 dr. (avoirdupois weight); the mean difference between the weights of the organ in the two sexes being 11 dr. From these observations all hearts weighing more than 12 oz. were rejected.

Bouillaud infers that the average weight is from 8 oz. 10 dr. to 9 oz. 11 dr.

Clendinning estimates the average weight, in persons from 20 to 60 years of age, of the male heart, at 8½ oz. avoirdupois; of the female, at 7⅖ oz. avoirdupois. Reid's observations give the average weight of the heart in males, from 25 to 55 years of age, as 11 oz. 1 dr., and in females as 9 oz.

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* "On the Weight and Dimensions of the Heart in Health and Disease." Reprinted from the "Monthly Journal of Medical Science," 1854.

2. The weight of the healthy heart is greater in persons who die after short periods of illness, and less in those who have suffered from emaciating or exhausting diseases. Taking this difference into account, the inference is, that in adult males, who have died from acute diseases, or from the effects of accidents, the ordinary weight of the heart is from 9 to 11 oz. avoirdupois, and in those who have died from chronic diseases from 8 to 10 oz. In females the ordinary weight of the heart, in acute cases, may be regarded as from 8 to 10 oz., and in chronic diseases from 7 to 9 oz. Occasionally the weight may fall as low as 5 or 6 oz., in persons of small frame, dying of exhausting diseases; and in powerful men, dying suddenly, or after a short illness, may reach 12 oz., or perhaps even more, without exceeding the limit of health.

3. The heart usually increases in weight with the advance in life. This is shown in the following table:—

Age.	Males.		Females.	
	Nos. Weighed.	Mean Weight.	Nos. Weighed.	Mean Weight.
		oz. dr.		oz. dr.
15 to 20	9	8 $2\frac{6}{9}$	9	8 $1\frac{6}{9}$
20 to 23	27	9 $0\frac{4}{27}$	21	8 $10\frac{9}{21}$
30 to 40	31	9 $7\frac{30}{31}$	19	8 $13\frac{16}{19}$
40 to 50	9	9 $11\frac{1}{9}$	5	9 3
50 to 60	15	9 12	6	9 $7\frac{1}{3}$
60 to 70	3	10 $13\frac{1}{3}$	—	—

It is not, however, very clear to what period of life this increase extends, and whether in very advanced age there is not, as is shown to be the case with the brain, a more or less marked decline in weight. These results agree with those obtained by Bizot, for the dimensions of the heart.

NORMAL DIMENSIONS OF THE HEART.

The following table, from Dr. Peacock,* gives the most important measurements of the healthy heart, according to the best observers:—

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* Dr. Peacock's measurements were taken from forty-two healthy organs, and were obtained as follows:—the aorta and pulmonary artery are cut across about an inch above their origin; the ventricles opened by incisions passing from near the aortic or pulmonary orifices to the apices; the auricles laid open transversely; without touching the valves or their attachments. The capacity of the orifices is ascertained by the passage of graduated balls, numbered from one to fifteen, and measuring from twenty-one to sixty-three lines. When the orifices and valves have been examined, the orifices are laid open and the length of the ventricular cavities measured from the attachment of the aortic and pulmonic valves to the apex. The thickness of the parietes is measured at three points, near the base where the walls begin to narrow; at the mid-

	M. Bizot.		Dr. Reid.		Dr. Ranking.		Dr. Peacock.	
	Males.	Females.	Males.	Females.	Males.	Females.	Males.	Females.
	In.	In.	In.	In.	In.	In.	In.	In.
Circumference of the heart ..	..	..	..	..	$9\frac{27}{28}$	$8\frac{13}{48}$	$9\frac{2}{24}$	$9\frac{7}{24}$
Growth of the right ventricle	..	..	..	..	..	..	$4\frac{22}{24}$	$5\frac{2}{24}$
,, left ,,	..	..	..	..	..	..	$4\frac{5}{24}$	$4\frac{7}{24}$
Length of cavity of right ventricle	$3\frac{18}{48}$	3	..	..	..	..	$3\frac{41}{48}$	$3\frac{43}{48}$
Length of cavity of left ventricle	$3\frac{2}{48}$	$2\frac{36}{48}$	..	..	..	..	$3\frac{16}{48}$	$3\frac{14}{48}$
Thickness of walls of right ventricle	$\frac{8}{48}$	$\frac{6}{48}$	$\frac{8}{48}$	$\frac{7}{48}$	$\frac{8}{48}$	$\frac{6}{48}$	$\frac{8}{48}$	$\frac{8}{48}$
Thickness of walls of left ventricle	$\frac{22}{48}$	$\frac{20}{48}$	$\frac{12}{24}$	$\frac{10}{24}$	$\frac{27}{48}$	$\frac{23}{48}$	$\frac{13}{24}$	$\frac{13}{24}$
Thickness of septum	$\frac{21}{48}$	$\frac{18}{48}$	$\frac{12}{24}$	$\frac{9}{24}$	$\frac{22}{43}$	$\frac{19}{48}$	$\frac{12}{24}$	$\frac{10}{24}$
Circumference of right auriculo-ventricular aperture	$4\frac{38}{48}$	$4\frac{13}{48}$	$5\frac{3}{10}$	$4\frac{9}{10}$	$4\frac{34}{48}$	$4\frac{8}{48}$	$4\frac{19}{24}$	$4\frac{13}{24}$
Circumference of pulmonic aperture	$2\frac{42}{48}$	$2\frac{31}{48}$	$3\frac{7}{10}$	$3\frac{5}{10}$	$2\frac{35}{48}$	$2\frac{24}{48}$	$3\frac{27}{48}$	$3\frac{26}{48}$
Circumference of left auriculo-ventricular aperture ..	$4\frac{1}{48}$	$3\frac{31}{48}$	$4\frac{6}{10}$	$4\frac{2}{10}$	$3\frac{45}{48}$	$3\frac{22}{48}$	$4\frac{1}{48}$	4
Circumference of aortic aperture	$2\frac{37}{48}$	$2\frac{26}{48}$	$3\frac{2}{10}$	3	$2\frac{31}{48}$	$2\frac{22}{48}$	$3\frac{7}{48}$	3

A progressive increase in the dimensions of the heart, from infancy upwards, had been demonstrated by Bizot, whose results are given in the following table. The increase of the heart, both in weight and size, with advanced age, is apparently a true hypertrophy; and is probably connected with the progressive rigidity of the arteries, which is a part of senile decay, and which must increase the arterial resistance. Also, up to a certain point, the weight of the body, and with this the mass of blood, increases. So that from both these causes the heart is called upon for more work, and thus becomes hypertrophied.

According to the following table, there is an uniform increase in all the dimensions of the heart, from infancy to old age, with one exception, viz., in females after the age of thirty, where there is a falling off in the thickness of the organ, the other dimensions continuing to increase. The heart appears, subsequently, to recover itself, and again to follow the general law, but not sufficiently to attain

point between the base and apex, and near the apex. (The middle measurement only is given here.) The septum is cut across half way between the base and apex, and measured across its centre. Dr. Sibson measures the orifices by means of a graduated cone, which is pushed through as far as it will go. Most observers simply measure the margin of the orifice after it is opened.

a thickness proportionate to that in the male heart of the same age. Mr. Bizot's measurements are taken vertically, from the apex to the base, round the base at the junction of the auricles and ventricles, and at the thickest part of the left ventricle.*

MALES.					FEMALES.				
Age.	Number of subjects examined.	Length of heart in Paris lines.	Width of heart in Paris lines.	Thickness of heart in Paris lines.	Age.	Number of subjects examined.	Length of heart in Paris lines.	Width of heart in Paris lines.	Thickness of heart in Paris lines.
1 to 4	7	$22\frac{4}{5}$	27	$10\frac{4}{5}$	1 to 4	8	$22\frac{5}{8}$	$25\frac{7}{8}$	$10\frac{1}{8}$
5 to 9	3	$31\frac{1}{5}$	33	$12\frac{4}{5}$	5 to 9	10	$26\frac{3}{5}$	29	$11\frac{3}{10}$
10 to 15	3	34	37	14	10 to 15	5	$29\frac{2}{5}$	$31\frac{1}{5}$	$12\frac{2}{5}$
16 to 29	18	$42\frac{5}{19}$	$45\frac{10}{19}$	$17\frac{17}{19}$	16 to 29	14	$38\frac{5}{7}$	$42\frac{9}{14}$	$17\frac{5}{7}$
30 to 49	23	$43\frac{3}{23}$	$47\frac{18}{23}$	$17\frac{4}{23}$	30 to 49	27	$41\frac{2}{27}$	$44\frac{1}{27}$	$14\frac{14}{27}$
50 to 79	19	$45\frac{12}{19}$	$52\frac{15}{19}$	$18\frac{5}{19}$	50 to 89	19	$42\frac{4}{19}$	$46\frac{11}{19}$	$16\frac{2}{19}$

The Paris line = ·083 of an English inch = 2·25 mm. The Paris inch = 1·065 English inch = 27 mm.

FORMS OF HYPERTROPHY.

Hypertrophy has been described as occurring in three forms, to which Bertin was the first to draw attention, and his classification has been adopted by subsequent writers. In the first, which is termed simple hypertrophy, the walls of the heart are thickened, while the cavities retain their normal dimensions; the second, excentric or aneurismal hypertrophy, presents an augmentation, both of the lumen of the cavities and of the substance of their parietes; and in the third, which has received the name of concentric hypertrophy, the former is reduced, while the latter is alone increased. The last variety probably has no existence as a morbid condition, but is, according to the showing of Cruveilhier and Dr. Budd, a post-mortem effect, an evidence, simply, of the powerful tonic contraction of a robust heart. The former writer observed that it occurred in almost all persons decapitated by the guillotine, and the latter has pointed out that in all concentrically hypertrophied hearts the ventricle may be easily dilated by means of the fingers, and always dilates of itself, when the rigor mortis goes off. The simple and excentric forms, then, are the two which alone constitute actual cardiac disease.

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* "Mémoires de la Société d'Observation," tom. i. p. 262.

The left ventricle in either form of hypertrophy is the part that is most frequently affected; next in order the right ventricle, and, lastly, longo intervallo, the auricles. There is not necessarily a relation between the increased thickness of the walls of a cavity and of the columnæ carneæ; the former may be themselves only thickened in some parts, while in others they retain their normal size; and again we occasionally find the trabeculæ much enlarged, while the proper walls present but little variation. Hypertrophy of the heart necessarily alters, more or less, the relation between the thoracic viscera, a point of importance to the practitioner, and also gives rise to modifications in the form and situation of the organ, differing according to the part which is most hypertrophied. Enlargement of the left ventricle from aortic disease produces elongation, and some increase of width. The apex beats lower and farther to the left than normal, and is formed entirely by the left ventricle. Hypertrophy of the same chamber from distant obstruction (or kidney disease) produces a more globular form of heart. The area of dulness on the front wall of the chest may be little, or not at all enlarged, but the girth of the heart is very greatly increased (to twelve or fourteen inches). In both these cases the lower lobe of the left lung will be found compressed, and in the former the liver will be depressed. Hypertrophy of the right ventricle (rarely if ever unaccompanied by dilatation) produces increase of the transverse diameter of the heart and extension of cardiac dulness to the right of the sternum. The apex of the heart may belong entirely to this ventricle in such cases.

In uncomplicated hypertrophy, where we have to deal with no morbid product but that of an increase in the amount of muscular fibre, the muscular tissue is of a deeper red than usual, and its consistency is increased; but the hypertrophy may be the result of a degenerative process, or a degeneration may have been set up in the organ, subsequent to the hypertrophy having been established; the colour may then be of a brownish tint, or present yellowish or fawn-coloured spots, while the consistency is generally increased, but sometimes reduced. In the former case we find the characters of voluntary muscular fibres more strongly marked than usual; the transverse striæ are more defined, and the edges have a sharper outline; in the latter these characters are more or less altered, and we meet with the further traces of the specific alteration. An analysis of the cases of fatty degeneration collected by Dr. Quain,* shows that the prevailing condition of the heart accompanying this state is one of hypertrophy, whether primary or secondary we are not prepared to determine; though it appears very probable that the change known as fatty degeneration is the result of various morbid processes inducing a disintegration of tissue. In the thirty-three cases of Dr. Quain's first series, the heart is stated to be enlarged in twenty-one; in six the organ was of a normal size; in four the dimensions are not stated; in one there was dilatation without hypertrophy, and only in one was the heart decidedly smaller than usual.

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* "Medico-Chir. Trans.," vol. xxxiii.

Perhaps more common than fatty degeneration in a hypertrophied heart is fibroid induration, presenting the characters before described.

Causes of Hypertrophy.—These are essentially of two kinds: "in the one kind," to use Dr. Watson's terms, "there is some mechanical obstruction to the exit of the blood from one or more of the cavities; a constricted state of the orifices is the most common condition. In the other kind, without any such mechanical drain or bar to the fluid, there is something to hinder the free and sufficient play of the organ, an adhering pericardium, it may be, or malposition of the heart. The causes of hypertrophy may, therefore, be situated within the heart itself, or without and beyond it; but in all those cases in which the effect of the hindrance or obstacle is to detain the blood in one or more chambers, the hypertrophy will be likely to be accompanied by dilatation, and generally speaking the hypertrophy and dilatation result from disease in some part, which lies beyond the affected chamber in the order of the circulation." The largest hypertrophied heart, however, which we have met with was one of the latter class, in which no such obstacle could be discovered. The specimen, which was taken from a middle-aged man, is preserved in the St. George's Hospital Museum; and weighed, when removed from the body, five pounds; the left ventricle is enormously hypertrophied, and very much dilated at the same time, but beyond this increase of size in the heart, no morbid appearances are perceptible or on record; the valves are all perfectly healthy.

In hypertrophy from obstruction, the causes will naturally be different for the two sides of the heart. Thus obstruction of the aorta, by adhesion or other affection of the valves, causes the left ventricle to work at greater tension, and at the same time keeps it constantly distended, so that it becomes both hypertrophied and enlarged. Aortic regurgitation by keeping a large quantity of blood in the chamber produces the same effect; and so, though not for the same reason, does mitral regurgitation. Again, simple hypertrophy without dilatation of the left ventricle, may be produced by obstruction in the distant parts of the arterial system; and this very frequently happens in cases of disease of the kidneys, a condition, which in some manner not yet quite explained, is connected with an increase of tension in the arteries. In so far as this is the case the effect is evidently analogous to that of immediate aortic obstruction. The increase of arterial tension which accompanies some forms of kidney disease, has been accounted for in several ways which we cannot discuss here; but no explanation is perfectly satisfactory, and hence some persons have supposed that the hypertrophy of the left ventricle and the other concomitant changes have no causal connection with one another, but are the effects of some general error of growth or development. This does not, however, explain those cases where hypertrophy is confined to the left ventricle; since a spontaneous developmental process would affect all parts alike. Aneurisms sometimes cause hypertrophy of the left ventricle, or at least accompany it.

The left auricle often participates in the enlargement of the ventricle, but hardly seems to be primarily affected, except in mitral obstruction, where it is first dilated and then hypertrophied.

Hypertrophy of the right side is far less common; partly because valvular affections are rarer on that side. Obstructions in the capillary circulation through the lungs are the most frequent cause: thus this form of hypertrophy occurs in chronic bronchitis, empyema, induration of the lung, &c. We may also point out a fact which we have not seen noticed elsewhere,—that curvature of the spine, whether angular or lateral, is often associated with hypertrophy of the right ventricle. This probably arises from the diminished capacity of the thorax, leading to imperfect restoration and occasional violent dyspnœa.

Obstruction or regurgitation at the mitral orifice also leads to dilatation, and sometimes hypertrophy of the right side, by hindering the flow of blood through the lungs.

An undeniable influence must be also attributed to inflammatory affections of the endo- and pericardium, as well as to chronic inflammatory conditions of the muscular tissue of the heart, producing fibroid patches. The manner in which pericarditis gives rise to it, is by causing partial or general adhesions, and thus preventing the free contraction of the muscular tissue. Endocarditis most commonly gives rise to hypertrophy, by inducing changes in the valvular apparatus, and thus affording impediments to the sanguineous current.

DILATATION OF THE HEART.

Hypertrophy is, as we have said, often accompanied by dilatation; but the latter condition may occur without any change in the thickness of the walls; or there may be actual thinning of them.

Hypertrophy with dilatation corresponds to the condition to which the older authors applied the term active dilatation; while dilatation, associated with a diminution of the fleshy parietes, was known as passive dilatation. The two conditions have also been respectively called, by Corvisart, active and passive aneurism of the heart. This latter term is however best reserved for another condition. We shall continue to use the term dilatation to designate a distinct class of morbid changes in the heart, and we shall separately consider the two varieties under which it occurs, the general and the partial form.

General dilatation without any attenuation is very rarely met with, though sometimes in a distended auricle the conditions may be very nearly balanced. Dilatation, with thinning of the walls, is the most common form, and seems to depend upon some degeneration or change producing weakness in the muscular substance. It is therefore essentially an atrophic condition.

The parietes of a dilated heart may be attenuated to an extreme degree; the thickest part of the left ventricle may be reduced to two lines in diameter, while at the apex the muscular substance may have disappeared entirely, so that the endocardium and pericardium are in apposition; at the same time, we find a corresponding diminution in the thickness of the columnæ. In one point there is a characteristic difference between hypertrophy and dilatation, independently of the nature of the lesion. This is in regard to the part affected. The left ventricle is most frequently attacked with hypertrophy, while we meet with dilatation most commonly in the right ventricle. It has been stated that the female sex are most prone to dilatation, and males to hypertrophy; we do not find this to be the case. On analyzing the ninety-two cases of heart-disease collected and reported in the "Medico-Chirurgical Transactions" by Dr. Barclay,* with a view to determining this question, we find that, of fourteen cases of dilatation, six occurred in females and eight in males; of sixteen cases of hypertrophy alone, seven were women and nine men; of forty-six cases of hypertrophy combined with dilatation, seventeen were females and twenty-nine males. On a rough average it therefore appears that in each variety there is a preponderance of about one-third on the side of the latter. Dilatation, as Dr. Hope remarks, takes place more in the transverse than in the longitudinal direction of the ventricles, and it accordingly communicates to the heart an unusually spherical form, the apex being rounded off in such a manner as frequently to be scarcely distinguishable.

Dilatation affects the auricles more frequently than hypertrophy; however, we must be careful in not hastily assuming a diseased condition, where its semblance is owing merely to distension. This is particularly the case with the right auricle, which very commonly appears much dilated, owing to the accumulation of blood taking place on this side of the heart in articulo mortis. If on removing the contents the cavity presents its normal appearance, we consider it to have been merely mechanically and temporarily distended; if the enlargement is persistent, we may look upon it as the result of morbid action during life. A dilatation of the right side of the heart is met with in connection with patency of the foramen ovale.

Partial Dilatation or Aneurism of the Heart.—Rokitansky assumes the existence of two distinct forms of cardiac aneurism. The first—the acute and rarer form—depends upon a laceration of the diseased endocardium, and adjoining muscular tissue, through which the blood passes; and the power of resistance being diminished at the point, a pouch is established, a fringed margin of endocardium is found at the entrance, and the blood deposits its fibrine within, while the margin becomes fringed with vegetations. Rokitansky has never seen a case in which the walls of an aneurism, formed in this manner, had become consolidated into a fibroid, callous tissue. In all the cases examined by him, the aneurismal formation was of recent date, having existed only a very inconsiderable period after the endocarditis, during the continuance of which it had originated. The second form is the more remote effect of an inflammatory condition of the investing or lining membrane, or of the muscular parietes of the organ. This induces the development of a fibroid tissue replacing, or, we should rather say, causing the absorption of, the muscular fibre. The new tissue contracts, the parietes lose their power of resisting the pressure of the blood, and a circumscribed dilatation ensues.

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* "Medico-Chir. Trans.," vol. xxxv.

The aneurism varies much in size, from that of a pea to that of the heart itself; it does not necessarily present an enlargement at the surface, nor does it appear to possess an inherent tendency to increase: thus in a case presented to the Pathological Society, by Dr. Jenner,* in 1848, an aneurism was found of the size of a filbert at the apex of the left ventricle. Though there was no muscular substance over the extremity of the sac, it did not project beyond the external wall of the ventricle. The existence of the aneurism in this instance was traced back to two years previous to the death of the individual. A few old slight adhesions binding the apex of the ventricle loosely to the free pericardium, indicated a previous inflammatory condition at the affected point. It appears that the healthy tissue of the organ possesses in many instances an inherent power of neutralizing the evil effects which might be expected to result from a local loss of resisting power in the parietes; otherwise it is difficult to understand how a cavity can be hollowed out in their substance without seriously impairing the contractile power. We sometimes find cases in which the thinning is not quite so definitely circumscribed as Rokitansky describes it, and where it yet proceeds to an extreme degree. Thus we have seen a case of enormous dilatation of both ventricles without hypertrophy, in which the apex of the left ventricle was thinned to the size of a sixpence (St. George's Museum, 1842-62). Partial aneurisms are not necessarily solitary, but there may be two or three, which may, as they progress, intercommunicate. Next to the apex of the left ventricle we find its base, and, third in order, the septum ventriculorum liable to be thus affected; in the latter case the bulging is towards the right side of the heart. Considering that there is a point in the upper part of the septum, at which normally there is no muscular tissue to maintain the separation of the two cavities, we should be led to expect the occurrence of aneurism more frequently at this point; but in ordinary circumstances the balance of the circulation suffices to prevent this result. When this form of aneurismal dilatation extends to such a degree that an opening and communication between the ventricles results, we have to deal with what Dr. Thurnam has termed the varicose aneurism; it is a condition analogous to a congenital state, not unfrequently met with, in which the ventricular septum is more or less defective.

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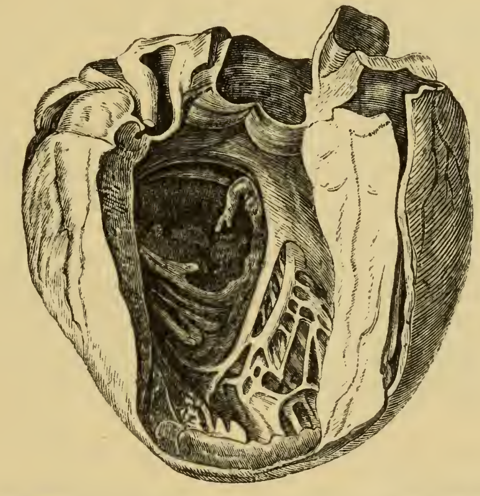
* "Report of the Pathological Society," 1848 9, p. 89.

FIG. 81.



Aneurism of the left ventricle, formed by dilatation of a circular portion of the anterior wall, in a girl aged nineteen.

The pouch was filled with a laminated coagulum; its mouth was narrow, round, and smooth, and its parietes consisting apparently of endo- and pericardium, with small deposits of a soft yellowish substance between them. The disease had probably commenced eighteeen months before death.

(St. Bartholomew's Museum. Series xii. No. 53.)

The contents of the cardiac aneurisms vary; we find in them fluid blood, more or less decolorized, sanguineous coagula, or laminated fibrinous deposits, resembling those found in arterial aneurisms, and presenting similar microscopic appearances. The walls may become ossified.*

The male sex and mature age offer a greater proneness to aneurism of the heart than the female sex and youth. The proportion with regard to sex is about 1 to 3. Hasse states that of forty-seven cases thirty-five were men and twelve women; and of forty-two cases, ten referred to individuals under thirty, and thirty-two to older persons.

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* See an interesting case described by Mr. Arnott, "Trans. Path. Soc.," 1868, vol. xix. p. 149, plate 7.

ATROPHY OF THE HEART.

In introducing the subject of dilatation, we observed that it was essentially an atrophic condition, Atrophy of the heart is also met with in the shape of a mere reduction of size, either as a result of wasting disease or as a congenital vice. Three extreme cases recorded by Burns probably belong to the latter variety; in one instance he found the heart of an adult as small as that of a new-born infant, and in another the heart of a female, aged twenty-six, was no larger than that of a child of six years. Bouillaud describes a case of a woman of sixty-one minutely, who died of acute peritonitis, in whom the heart was a third smaller than in the normal condition, or about the size of the heart in a child of ten or twelve. The surface was furrowed and presented milk spots, the remains of former pericarditis; the cavity of the left ventricle was scarce large enough to contain a pigeon's egg, and its parietes were only three lines in thickness. In phthisis there is a uniform diminution in the size of the heart, as illustrated by the following measurements given by Bizot * of the heart in the adult:—

Males.				Females.			
Age 16 to 79 years.	Length of heart lines.	Circumference of heart lines.	Thickness of heart lines.	Age 16 to 89 years.	Length of heart lines.	Circumference] of heart lines.	Thickness of heart lines.
57 Phthisical ..	$42\frac{7}{12}$	$47\frac{1}{24}$	$15\frac{11}{12}$	—	$39\frac{3}{5}$	$41\frac{1}{30}$	$15\frac{1}{13}$
65 Non-phthisical	$45\frac{1}{31}$	$50\frac{28}{31}$	$18\frac{13}{31}$	—	43	$47\frac{14}{16}$	$16\frac{9}{28}$

A heart from a woman, aged forty-seven, which weighed only three ounces one drachm, was shown to the Pathological Society.† The patient had died of inanition, from pyloric obstruction.

Dr. Peacock finds that the weight of the heart in persons who have died of phthisis is less than in those who have sunk from other diseases; but the decrease of weight from that cause is not so marked as in persons who have died from other chronic affections, unconnected with disease of the lungs.

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* "Mémoires de la Société d'Observation," vol. i. p 277.

† Church: "Trans. Path. Soc.,' 1868, vol. xix. p. 147.

ADVENTITIOUS PRODUCTS.

The new growths met with in the heart substance are fibrous or connective tissue masses, tubercle, cancer, and syphilitic growths. Cystic parasites also occur.

New growths of connective tissue sometimes occur in a discrete form as well as in the form of general induration already described. Several such cases are recorded. In one of which an account is given in the Transactions of the Pathological Society, sudden death occurred in a man supposed to be perfectly healthy, and the heart was found to contain numerous masses of fibrous tissue replacing the muscular tissue.* In this, as in other cases,† it has been suggested that such growths are syphilitic; but positive evidence has always been wanting. Undoubted syphilitic growths have been observed, they may exceed a hazel nut in size, and in a case recorded by Oppolzer, appeared to cause rupture of the heart. They may also, as already stated, lead to suppuration.

Tubercle is very rarely observed in the heart; when it occurs it is usually associated with tubercle of the pericardium, in cases of general tuberculosis.

Cancerous growths are not common; the best account of them is to be found in a paper by Dr. Peacock,‡ in which he has analyzed forty-five recorded cases of the disease. As the older accounts are defective in point of histology, it is probable that many of these would not now be described as cancer. Only two of these were cases of primary cancer of the heart without participation of any other organ. In five more the disease appeared to occupy other organs simultaneously, while in eight it spread to the heart from adjacent parts, such as the mediastinal glands. This form of growth is less uncommon than the former; it often comes under the anatomical description of sarcoma rather than cancer. In about half the cases the disease was secondary to cancerous growth elsewhere. The growth assumed the form of "cancerous infiltration" in a few instances, but in the majority there were distinct tumours. The seat of disease was generally the right auricle and ventricle, either alone or in conjunction with the left auricle and ventricle. The form of cancer is described as being encephaloid in about half the cases; scirrhus and melanosis occurred in a few, while in others the species of cancer is not distinguished.

Ossific deposits are recorded, but they seem invariably to proceed from the endocardium; and we shall return to the subject in connection with the diseases of this membrane. Both Corvisart and Hope give cases of portions of the heart being converted into cartilage; but there is little doubt that if these cases had been subjected to microscopic examination, the tissue would have proved fibrous rather than cartilaginous.

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* Dr. Whipham: "Trans. Path. Soc.," 1870, vol. xxi. p. 115

† "Trans. Path. Soc.," 1868, vol. xix. p. 108, plate iv., figs. 4, 5, and 6.

‡ "Trans. Path. Soc.," 1865, vol. xvi. p. 99.

Parasites are very uncommon, but cysticercus and echinococcus-cysts have occurred. A well-marked instance of the latter was presented to the Pathological Society by Mr. Ward in 1847, and the preparation is still preserved in the Museum of the London Hospital.* In this case no trace of the entozoon was discovered in other viscera. A remarkable case in which a cyst containing hydatids was found in the substance of the heart, is recorded and delineated by Mr. Evans in the seventeenth volume of the Medico-Chirurgical Transactions. It occurred in an unmarried female, about forty years of age, who, during the winter preceding her death, had been subject to palpitation and angina pectoris. The cavity of the pericardium was found to be coated with a layer of coagulable lymph over a small extent of its front surface; the apex of the heart was lost in a considerable tumour, apparently an elongation of the heart itself, and covered on all sides by pericardium. The new growth was found to project into the cavity of the right ventricle, was smooth, globular, and about three inches in diameter. It contained numerous hydatids from the size of a pea to a pigeon's egg; their interstices being filled up by a soft, curd-like, yellow substance. The hydatids were precisely the same as those found in the liver. The trichina spiralis has not been met with in the heart.

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* "Report of Pathological Society," 1847-8, p. 225.

CHAPTER XX.

MORBID ANATOMY OF THE ENDOCARDIUM.

It has required the multiplied observations of numerous labourers in the field of pathology to establish the true nature and importance of endocardial affections: Laennec and J. P. Frank were among the first to draw attention to inflammatory conditions of the endocardium, but we owe the more correct appreciation of the subject to Bouillaud and Hope. The pathology of the endocardium is as significant in regard to its primary, as it is with regard to its secondary, lesions. The membrane is analogous to that lining the blood-vessels; it consists of a layer of epithelium, between which and the muscular fibre of the heart there is a layer of connective tissue; it is in the latter that blood-vessels ramify, and through them the nutrition of the surface-laminæ becomes affected, in disease as well as by the direct influence of the blood contained in the cavities of the heart. It is necessary to bear this double relation in mind, as, without a due perception of these facts, we shall find it difficult to harmonize our general knowledge of morbid changes with the apparent exceptions that endocardial disease brings to our notice.

A careful examination of the endocardium is necessary, before we determine positively that the appearances we find are due to pathological alterations; the earlier observations lose much of their value from post-mortem staining having been mistaken for inflammatory reddening. When the change of colour, which, naturally, is of the palest white and translucent, is due to inflammation, the redness cannot be washed off, being, in fact, situated beneath the epithelium, nor is it rapidly destroyed by maceration; the membrane is pulpy and thickened, and in an advanced state, further products of inflammation are found beneath or upon the membrane. Where the redness is the result of mere imbibition, we usually find the blood in the cavity fluid. The later the post-mortem examination is made after death, and the warmer the weather at the time, the more likely are we to find the lining membrane of the heart and arteries stained with blood. The redness of imbibition is darker in proportion to the period the blood is in contact with the parietes of a cavity, and for this reason it is observed, as pointed out by Hasse,* in the following descending order: darkest in the right auricle, paler in the right ventricle, with the exception of the valves of the pulmonary artery, which are as deeply coloured as the auricle; still paler in the left auricle, whilst the left ventricle often retains quite its natural tint, except that the aortic valves are darker. In the great vessels, the posterior surface is strikingly dark, in comparison with the anterior.

We rarely have an opportunity of seeing endocarditis in its first stage. Rokitansky and Hope describe inflammatory redness of the endocardium as mottled. Dr. Hope states it to be less characterized by streaks, patches, isolated unstained spots, and abrupt edges, than non-inflammatory imbibition. Another change is that of opacity of the lining membrane. This alteration in the appearance of the endocardium also changes it in other respects, it loses its glaze and becomes dull, relaxed, milky, and velvety.

In rare cases, an adhesion has been found effected between opposite points of the parietes. When the inflammatory process extends to the valves, the consequent change in their relation to the blood current, gives rise to those physical signs which are of so much value in the diagnosis of cardiac affection.

It has been supposed that inflammation of the endocardium must be like that of the pericardium or other serous membranes, accompanied by the production or "exudation" of lymph on its free surface; and that it is rarely or never to be found here only because the current of blood constantly washes it away. On the other hand, it has been said that the endocardium itself is without vessels, being nourished by the current of blood within its cavity, and therefore exudation, in the ordinary sense, is impossible. Some authorities have carried this objection so far as to maintain that inflammation of the endocardium is itself impossible. This question has led to a great deal of controversy into which we cannot enter here; but it may be laid down that no exudation of fibrin or production of inflammatory lymph on the endocardial surface has ever been proved to take place; while on the other hand changes are observed in the endocardium, to which no other name than that of inflammation can be applied. It has now been shown that there is beside the inflammation of the sub-endocardial structures which produces the phenomena already described, a multiplication of the connective-tissue cells of the inner coat, which gives rise to the appearance of a deposit or exudation. Endocarditis affects the valves far more commonly than any other part; so much so, that when we speak of endocarditis we generally mean an affection of the valves.

The results of endocardial inflammation are frequently observed in the aortic and mitral valves as a consequence of rheumatism.

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* "An Anatomical Description of the Diseases of the Organs of Circulation," Syd. Soc. Ed., p. 128.

They are characterized by a row or fringe of translucent nodules along the line of apposition of the valves. When quite recent they have a close resemblance to the normal corpora Arantii seen on the semilunar valves; but are very numerous. These nodules or beads are now known to be formed in the first place by a hyperplastic growth of the connective tissue of the valve itself; they readily cause the precipitation upon them of fibrin from the blood, and the new growths thus enlarged form the villous excrescences which are called vegetations. These will be further considered below.

Suppuration.—If the presence of lymph on the inner surface of the heart has never been satisfactorily established, it follows à fortiori, that we still less frequently meet with a purulent effusion resulting from endocarditis. The loosening of the tissue, the want of polish, and the felt-like character of the endocardium, are sometimes very strongly marked in the centre of inflammation, and hence lacerations frequently occur. In these cases a purulent product mixed with the blood may be found infiltrated into the tissue, if not at the surface of the endocardium, whilst abscesses are occasionally found to have spread themselves over a various extent of surface below the endocardium, in the cellular and adjoining muscular strata, deep in the tendons and in the tissue of the valves.

Results of Endocarditis.—The most important of these are—(1) Ulceration, (2) Fibrous thickening, (3) Dilatation, and (4) Embolism in distant parts. One of the effects of endocarditis is ulceration; it is met with on the parietes of the heart, but more frequently on the flaps of the valves, where it gives rise to perforation and rupture. Perforation of the septum ventriculorum occasionally results from this cause.* A free communication may thus be established, or the muscular wall may be completely destroyed, but an exudation of fibrine having taken place on the distal side, the intermingling of the contents of the two cavities be prevented. Ulceration and suppuration commonly give rise to pyæmia, which is due both to the mingling with the blood of some fever-producing substance and to the blocking of distant capillaries or small arteries with masses detached from the heart.

Chronic ulcerative endocarditis is a form which deserves to be specially distinguished, since it may during life produce the symptoms merely of a chronic pyæmia, such as repeated rigors, high temperature, and what are called "typhoid" symptoms. It commonly occurs on the left side of the heart, but we have seen one case affecting the tricuspid valve, and causing obstruction of branches of the pulmonary artery with hæmorrhagic blocks.

It is, however, sometimes difficult or impossible to say whether the heart affection is the cause or the consequence of the general blood-poisoning,* and there is no doubt that the latter is sometimes the case,† true suppuration of the valves being found.

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* "Trans. Path. Soc.," vol. vi. p. 143.

Other results of endocarditis are thickening and induration. Sometimes the whole inner membrane of one or more cavities is white and opaque, a phenomenon which is set down to chronic inflammation. In other cases limited patches of induration and thickening are seen, about which it may be doubtful whether they are evidence of a chronic change, or an acute process which happened some time previously. Rokitansky describes white patches, under the name of tendinous spots, as a sort of cicatrices resulting from inflammation of the endocardium and adjacent parts of the muscular tissue. These patches are often seen in the upper part of the wall of the left ventricle, near the aortic valves, and especially in cases of aortic regurgitation. They are sometimes membranous and not unlike valves. It has been thought that friction of the regurgitant stream of blood might have something to do with their causation, but the explanation is plainly inadequate. Their production appears to depend upon two circumstances: first, upon a general chronic inflammation by which the inner coat becomes œdematous and partly detached from the subjacent tissue so as to be freely moveable upon it; and secondly, upon the action of the regurgitant stream of blood throwing the loosely attached membrane into ripples or folds, which finally become rigid and thus form membranous expansions or fibrous ridges. These surfaces are sometimes shaggy or covered with papillary vegetations.

Dilatation of the heart is also a result of endocarditis, though perhaps not unless the muscular substance participate in the inflammation. It may be partial, producing a cardiac aneurism or a general enlargement of a whole cavity.

Embolism, or the blocking up of vessels in distant parts of the arterial system, with particles detached from the heart, is another result of endocarditis. There may be arterial or capillary embolism. In this way are produced the so-called "fibrinous blocks" of the spleen and kidneys, and more rarely similar infarctus in other parts, as in the brain. Very rarely endocarditis of the right side produces similar changes in the lungs.‡

Endocarditis is far more common on the left than on the right side of the heart; and the latter is still more rarely affected without the former. On the other hand, congenital affections, or so-called malformations of the heart, which are doubtless sometimes produced by endocarditis, are more frequent on the right than on the left side.

Coagulation of Blood within the Heart.—A frequent concomitant of endocarditis, according to Bouillaud, is the coagulation, during life, of the blood, and the organization, in the clot, of new blood-vessels. This occurrence is now believed to depend not on endocarditis, but on other causes imperfectly understood. It is, however, not the less important. The coagula formed by inflammation were described by Bouillaud as colourless, elastic, and glutinous, and closely resembling the buffy coat of inflammation, or false membranes themselves. The symptom by which Bouillaud recognized this occurrence before death was a want of accordance between the pulse and the heart in point of force; the heart presenting evidence of violent excitement and action, while the pulse was small and evanescent. Gluge* describes organized fibrinous coagula under the name of hæmatoma, and gives an interesting description, with the minute anatomy of the clot, of a case which occurred in a female, aged fifty-two. The left auricle was filled with a red tumour, surrounded by a delicate membrane, in the interior of which he distinctly traced capillary vessels, forming a retiform plexus. Similar instances may be also found in the records of the Pathological Society, and in Dr. Hodgkin's Catalogue of Guy's Hospital Museum.

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* "Trans. Path. Soc.," vi. 1855, p. 151.

† Dickinson, "Trans. Path. Soc.," xvii. 1866. p. 76, records two such cases.

‡ The subject is further discussed in the chapter on Embolism.

The surface in such cases is found more or less intimately connected with the endocardium, while the interior of the clot may, in its turn, be undergoing further changes of an inflammatory or degenerative character. The fibrine is seen to be breaking up into a granular condition; exudation or inflammation corpuscles and fibro-plastic cells may be exhibited by the microscope. This sometimes gives rise to a substance almost exactly resembling pus to the naked eye, but the microscope will show well-marked differences. The elements seen are mostly atrophic and degenerated leucocytes, and even if cells which individually resemble pus-cells are found, they are not in so great numbers as to constitute pus. Sometimes the clot is converted into a mere bag filled with puriform material. These coagulations very often assume a globular form, constituting the *végétations globuleuses* of Laennec.

Distinction of Ante-mortem Coagula.—The older pathologists attributed a much greater importance to fibrinous coagula, or, as they termed them, polypi, in the heart, than they now obtain, owing to their being regarded as the immediate cause of death. It is only in rare cases that we shall be justified in looking upon them as products formed during life; in the majority of instances they are merely the first evidence of the arrest of vitality, and the incipient influences of the metamorphoses of decay. When formed during the agony, or after death, there is no adhesion to the parietes; the polypus is moulded to the cavity which contains it, and a straw-coloured fibrinous layer invests a blood clot, similar to the buffy coat covering the coagulum of blood obtained by venesection. The clearest distinction is, however, afforded by the manner in which the constituents of the clot itself are arranged. If the red corpuscles are uniformly distributed through the fibrinous network, so that the whole resembles an ordinary clot formed out of the body, its appearance supplies no reason for thinking that it was formed before death. If, on the other hand, the constituents are separated, we must look at the manner in which they are arranged. Supposing that there is a layer of yellow fibrine in that part which (in the position of the body after death) is uppermost, and that the lower parts contain red corpuscles, the arrangement is evidently the work of gravitation, as in the buffy coat which sometimes forms in the bleeding basin after venesection; and there is no ground for supposing the coagulation to have been ante-mortem. On the other hand, we sometimes find white and yellow masses, composed of fibrin alone, while the coloured elements of the blood are found in other parts, where their distribution is not to be explained by the law of gravitation; and in these cases we are justified in concluding that the separation of fibrin took place while the heart was still in motion (though perhaps feebly), so that the coloured parts were propelled away from the gradually depositing mass of fibrin. The conclusion will be strengthened if the fibrin be found adherent to the walls. In other cases we can have no evidence that coagula were formed during life, unless they have existed long enough for secondary changes to have taken place in their interior, such as organization, formation of blood-vessels, or the softening which resembles suppuration, above described. In these respects they are strictly comparable to thrombi in veins. Another important point is the position of the clots. When coagulation takes place in consequence of retardation of the blood current during life, it will always be in those parts where the blood current is naturally slowest; *i.e.*, the apices of the ventricles and the appendages of the auricles—and, as it seems, most frequently, the appendage of the right auricle and the apex of the left ventricle, among the muscular trabeculæ. From these points they may, by the deposition of successive layers, spread through the whole of the cavity. It should also be remembered that any roughness or alteration of the endocardium, as of the inner coat of vessels, will cause coagulation on its surface, a perfectly normal condition of the walls of blood-vessels being, according to the researches of Brücke, a most important factor in preserving the normal fluidity of the blood.

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* "Atlas der Pathologischen Anatomie," Lieferung 11.

CHAPTER XXI.

DISEASES OF THE VALVES OF THE HEART.

THE estimates of different authors with regard to the influence exerted by inflammation in producing valvular disease, have varied considerably. Bouillaud attributes nearly all changes occurring in the valves, such as altered consistency and form, fibrinous concretions, calcareous and ossific deposits, to inflammatory action, terming them the third stage of the inflammatory process; while Rokitansky, and many with him, are of opinion that they are only in part the product of endocarditis, but that the majority are the result of slow changes of nutrition, not connected with inflammatory action. The most manifest direct results of endocarditis are white opacity and thickening of the endocardium and the lining membrane of the valves, and adhesion between the latter. These adhesions are most commonly found in the aortic valves, and this lesion must necessarily constitute a permanent and very serious obstacle to the circulation, in its turn giving rise to further disorganization and derangement, such as hypertrophy and dilatation, dyspnœa and anasarca. In dealing with this species of malformation it is often very difficult to determine whether it is congenital, or the result of disease, especially when, subsequent to adhesion, an absorption of the partition separating the two pouches is effected, and the double valve thus converted into one. The varieties which the fusion of the valves with one another, or their adhesion to the walls of the heart, may present, are very numerous. In all cases an insufficiency of the valves must result, which both offers an obstacle to the free discharge of the blood from the heart, and fails adequately to close the orifice during the diastole, so as to prevent regurgitation. The left side of the heart generally, and especially in regard to inflammation, offers by far the greatest proclivity to disease. Very few cases are recorded in which a phlogistic process could be demonstrated on the right side. Gluge * gives two observations in which the tricuspid valve was thickened and rendered insufficient by this cause. We also find two instances reported in Dr. Hodgkin's Catalogue of the Museum of Guy's

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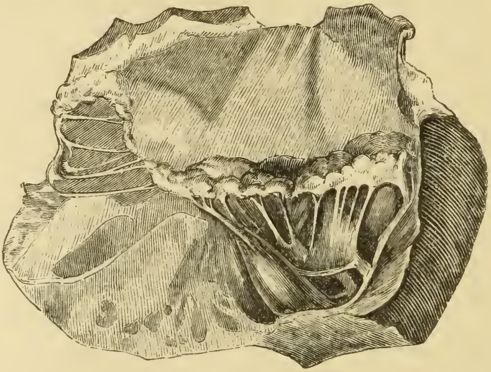
* "Atlas der Pathologischen Anatomie," 1850, Lieferung i. Beobachtung, 12 and 12a.

Hospital (Nos. 1401 and 1402), in which the curtains of the tricuspid were thickened. In one of these there was also shortening of the tendinous cords. In the Museum of St. Mary's Hospital is a specimen of adhesion and thickening of the tricuspid valves, with contraction of the orifice apparently the result of inflammation. We have also seen one case of ulcerative endocarditis affecting the tricuspid valve. In both the cases just mentioned the left side of the heart was quite normal. The valves of the pulmonary artery are also little liable to disease; most of the affections of this orifice being congenital.

Thickening of the Valves.—While the arterial valves are more subject to adhesion, we find the mitral valve more prone to an hypertrophy of its fibrous tissue, which is especially liable to present itself in the shape of nodulated masses, or granulations, marking the line of apposition of the flaps, and in some instances closely resembling accumulations of fat. In connection with hypertrophy of the endocardium we find the lining membrane of the valves also thickened, by which means it appears that, independently of inflammatory action, a secondary adhesion may be effected between the flaps. To these chronic changes the aortic valves are more liable, though it is not at all unusual to find the curtains of the left auriculo-ventricular orifice opaque throughout, from the same cause.

Fig. 82.



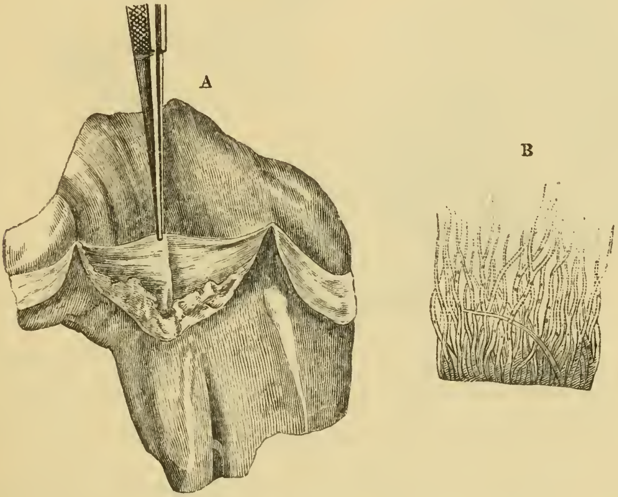
Fibroid thickening of the mitral valve.

The process which gives rise to thickening and to adhesions is probably always the same, and consists essentially in an increase of the connective tissue of the valves themselves, similar to that which gives rise to the granulations of acute endocarditis. In recent cases the new growth consists of tissue with a great abundance of nuclei; and in later stages are seen spindle-shaped cells, and an increase of fibres. This newly-formed tissue is particularly liable to degeneration; in the first place to a granular metamorphosis, not unlike some of the changes referred to atheroma in arteries, and also to calcareous degeneration, producing the calcification or so-called ossification of the valves spoken of further on. These changes may affect either the fibroid tissue resulting from acute endocarditis (as in rheumatism) or that produced by the more chronic changes which are sometimes called chronic inflammation; and hence it is difficult or impossible to say, from their appearance alone, whether the morbid changes of valves are due to an acute or to a chronic disease.

Opacity of the valves usually results from the chronic changes just described; and it often happens, as in atheroma of arteries, that the new growth is calcified. Some degree of opacity is very common, especially in the anterior flap of the mitral, where a yellow opaque patch is the rule in persons beyond middle life. It is not impossible that this is produced by friction, as this flap appears often to touch the ventricular septum.*

Atrophy of Valves.—We have already alluded to perforation of the valves, as a result of endocarditis. Another form in which the same lesion occurs is in connection with atrophy. This is manifested, in the first instance, by attenuation and increased transparency of the valves; as this advances, one or more openings are effected, which may be sufficiently numerous to induce a cribriform appearance. It is only when the perforations are large or numerous that they interfere, to any serious extent, with the circulation. Dr. Wilks is of opinion that most of these perfora-

FIG. 83.



Fibroid thickening of a pulmonary valve, extending symmetrically on both sides of the curtain, and consisting of a soft fibrillating deposit.
It was found in a man who had a broken spine.

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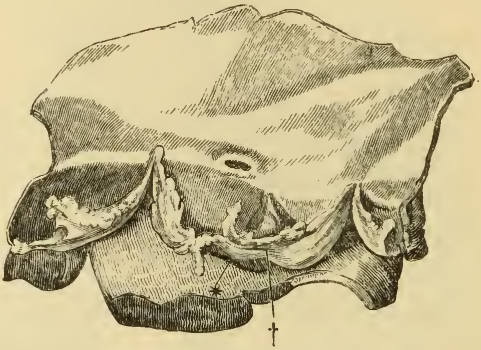
* Dr. Moxon has suggested that the white fibroid patches on the ventricular wall may be due to the same friction, but they are far less common than the corresponding affection of the valve.

tions are congenital. Dr. Kingston,* who was the first to draw attention to this point, observes that atrophy may be defined as a simple shortening of the valve, and, in the first instance, a mere atrophy in the direction of the length. He speaks of the cribriform appearance in the flaps as also resulting from the same process, and has found the two conditions chiefly in the mitral and tricuspid valves. In this he differs from other authors. Rokitansky, for instance, has only met with the lesion in the arterial valves. Dr. Kingston, out of about thirty cases of valvular disease, found the lesion to be atrophy in ten. The mitral valve was shortened in five, the tricuspid in five; both in two. In one the mitral valve was cribriform, in two the tricuspid, and in one both the aortic and pulmonary valves were so.

Rupture of the Valves.—It is doubtful whether perfectly healthy valves can be ruptured by violent or excessive tension, but there can be no doubt that this accident often occurs in valves weakened by disease.† The causes are usually great muscular exertion, and vomiting or sudden mental shock. Rigidity or brittleness arising from calcareous deposit is the most frequent predisposing cause; but it is said that a soft gelatinous condition may lead to lacerations, especially of the valves of the aorta. The fissured appearance of the true rents thus produced distinguishes them from the perforations resulting from atrophy.

Fig. 84.



Atheromatous deposit in the valves of the aorta of a man aged twenty-six, with rupture at the point marked by *; there was also congenital union at the point marked by † of two of the valves.

The case is described in the "Reports of the Pathological Society," vol. iv. p. 100.

Calcareous and ossific deposits, which we have seen to be not uncommon on the surface of the heart, are very rarely met with under the endocardium, except in connection with the valves. They present the most varied forms, which may be compared to the fantastic shapes assumed by molten lead when poured into water; sometimes resembling stalactitic projections, at others forming irregular rounded eminences, stretching across the orifices of the heart like rigid bars, maintaining the valves in a state of permanent erection or distension, and inducing symptoms both of obstruction and of regurgitation. A single flap or curtain may be rendered rigid while the others retain their natural pliability: the valves of one side of the heart may be more or less intimately united by the morbid growth; but whatever forms the lesion may assume, it is scarcely possible to occur without a permanent narrowing of the orifice. They are, as has been said, always connected either with those chronic changes of the endocardium, which may be called slow inflammation, or else are a result of antecedent acute inflammation. Kreissig and Bouillaud refer the disease uniformly to inflammatory action; and Dr. Watson is also of opinion that it is somehow certainly connected with inflammation of the internal lining of the heart. But we must not overlook the important fact of the natural tendency existing in the arterial system generally, as well as in other tissues of the body, to induration and ossification with advancing life; and though we are far from looking upon ossification of the valves as a physiological process, we are justified by analogy, as well as by the positive fact of the very chronic nature of these deposits, in looking upon them in many cases as of a non-inflammatory character allied to the general species of degenerative disease. At all events, they must fall under the same category as atheromatous and calcareous changes in the arteries.

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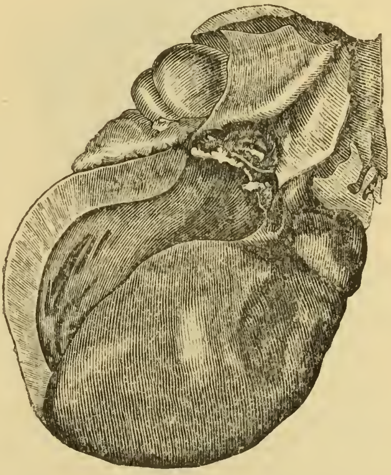
* "Medico-Chirurgical Trans.," vol. xx. p. 90.

† See "Trans. Path. Soc.," 1853, vol. iv. p. 100.

Fig. 85.

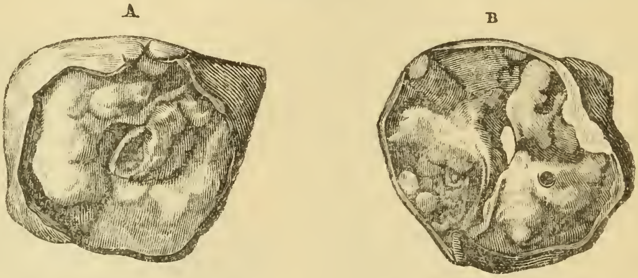


Aortic valves of a man aged forty-seven, rendered perfectly rigid by calcareous deposit.

The patient was affected with granular kidneys and cirrhosis of the liver.

Although we have used the term ossification in accordance with common usage, to designate the change under consideration, it is important not to confound the process with the one in which genuine bone is formed; cretification or calcareous deposition would be a more appropriate term, for there is no resemblance between the morbid product and true bone. It consists essentially of carbonate and phosphate of lime deposited in irregular, amorphous nodules, and resembling more a chemical precipitation than an organic formation. The material is more or less friable, and is connected by the remains of the fibroid, or atheromatous matter, in which it formed. It is soluble in the mineral acids. It is often difficult, when we meet with an advanced case, to determine in what part the deposit first takes place; whether beneath or on the surface of the lining membrane. The opinions of different writers differ with regard to this question. The most common form undoubtedly is the conversion of atheromatous or fibroid deposit underneath the lining membrane analogous to what we see occurring in the arteries; and as this enlarges, the membrane becomes softened and destroyed, and the ossification then projects free into the sanguineous current. It is not the mere increase of the deposit which determines this solution, but an element in producing this result is undoubtedly a morbid affection of the lining membrane itself, in which, even in early stages of degeneration of the subjacent tissue, we have observed disintegrating processes, of which we shall speak further when discussing the diseased conditions of the arterial system. One of the most extreme cases of narrowing of the aortic orifice in an adult, that we have met with, is the one delineated (Fig. 86), in which the continuity of the lining membrane was preserved entire over the ossific deposit. The passage was contracted to the size of a pea.

Fig. 86.



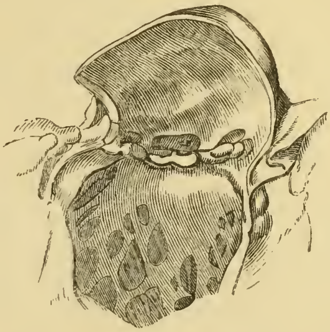
Ossification of the aortic valves; a thick calcareous deposit has taken place between the valvular membranes, interposing a rigid and almost imperforate diaphragm between the cavity of the heart and the vessel.
A, Upper surface; B, under surface.
(From St. George's Hospital Museum, E 18.)

Rokitansky points out that there may also be a calcareous metamorphosis of the fibrin, which is coagulated on the valves in the form of vegetations. This change gives rise, he says, to stalactitic osseous masses, which occasion and promote the continued formation of new vegetations; and are consequently very commonly surrounded by them. Calcareous concretions and morbid affections of the valves generally follow the law which determines the great prevalence of disease on the left side of the heart as compared with the right side: ossification especially is so rare on the right side that it has been denied altogether. Hasse, however, has seen partial ossification of the pulmonary artery; and Dr. Hodgkin* also reports a case of thickening and bony deposit in the pulmonary artery close to the valves.

* "Catalogue of Guy's Hospital Museum," No. 1403.

Combined Lesions.—The lesions of the valves hitherto spoken of may be variously complicated with one another, or with heterologous growths. Pathological records contain instances of a great variety of changes of form, the result of morbid processes or accident. Thus the individual flap of the semilunar valve may be reverted or inverted, the valves of the aorta may become detached at their bases, and thus lose the fulcrum by which they resist the impetus of the blood; or the tendinous cords of the mitral may induce a deficiency of the valve by a shortening and thickening, a lesion which Hope considers as constituting one of the worst varieties of diseases of the valves.

Fig. 87.

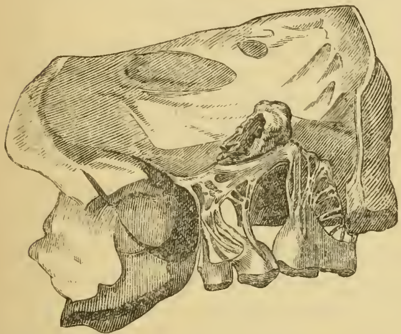


Aortic valves of a child aged four years; they are opaque and thickened, and their free margin curled backward toward the artery. Two of the valves are closely united by their adjacent margins.

(St. Bartholomew's Museum. 11th Series, 52.)

Aneurism of the Valves.—A condition of the valves remains to be pointed out, which was first demonstrated by Dr. Thurnam;* it consists in a saccular dilatation, which he attributes to a gradual distension, and hence terms aneurism of the valves. It is met with in the aortic and tricuspid, but most commonly in the mitral, valves. Dr. Peacock† has also recorded a similar affection of the valve of the foramen ovale. We find that the dilatation may exist without any lesion of continuity in the tissue; the endocardial lining being traceable throughout the pouch. This in the case of the mitral valve projects into the left auricle, and is often filled with a clot of blood. This form would correspond with what is termed true aneurism of the arteries. A second variety is that resulting from inflammation of the valves, by which a solution of continuity is effected in the lining membrane of one surface of the flap. Blood penetrates by this opening between the two layers of

Fig. 88.



Aneurism of the mitral valve; a pouch projecting into the cavity of the left auricle, about three-quarters of an inch high, and half an inch wide. It has burst by an irregular rent on one side.

(St. Bartholomew's Museum, 12th Series, 62.)

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* "Medico-Chirurg. Transactions," vol. xix. p. 162, vol. xxi. p. 187, vol. xxiii. p. 323.

† "Pathological Reports," 1850-51. p. 80. Several instances of valvular aneurism are detailed in the same volume, pp. 72, 77, and 78.

endocardium, causing that of the opposite side to bulge out, forming, in fact, a sort of dissecting aneurism. This form is described by Rokitansky and Förster as occurring only on the left side of the heart, and attaining the size of a hazel nut, or even of a walnut. It is usually formed rapidly, and often becomes ruptured, leaving a ragged opening thickly beset with vegetations. This species of aneurism has been described as a hernia-like protrusion of the valve.* Another characteristic example is described as an aneurism by Dr. Andrew.†

Causes and Results of Valvular Disease.—We have, for the sake of convenience, reviewed the diseases affecting the individual tissues of the heart separately; but before proceeding further, it may be well to dwell for a brief space upon their complications with one another, and with morbid phenomena in other vital organs. The fact of the intimate connection between a rheumatic diathesis and pericardial and endocardial inflammation has already been alluded to. We cannot show the relation better than by extracting from Dr. Latham's "Lectures on Clinical Medicine" the statistical facts illustrative of the subject, to which that author's large experience had led:—The number of cases of acute rheumatism which occurred to him were 136, out of which 90 presented symptoms of heart disease; of these 63 were diagnosed as affecting the endocardium alone, 7 the pericardium alone, and 11 both endo- and pericardium. Out of the total number only three proved fatal; they were men, and in them both surfaces of the heart were inflamed. In all cases of heart disease other organs will be liable to be affected in proportion, as different parts of the circulation are more immediately involved. While disorders of the arterial system more directly induce deranged action in the brain, the spleen, and the kidneys; the lungs, the liver, and the chylopoietic viscera suffer chiefly in derangements acting immediately upon the venous system. As a matter of course, this distinction is one that cannot be always demonstrated, as in an advanced stage of cardiac disease of either side of the heart, or of any one portion, the entire circulation must of necessity be impaired. On the arterial side we find that more particularly a complication between granular degeneration of the kidneys with heart disease obtains; thus Dr. Bright has shown, that in a hundred cases of this disease, the heart presented lesions in at least thirty-five, a number which would probably have been increased if the condition of this organ had been noted with the same care in all. This proportion has been confirmed by the researches of Dr. Taylor.‡ The secondary effects produced by the dislocation of coagula from the left side of the heart, in the brain, the spleen, and the kidneys, by blocking up the arteries, and thus altering the nutrition of the parts to which they lead, will be spoken of hereafter. The influence of valvular disease in producing hypertrophy is a point of great importance, and its connection with pericardial and endocardial inflammation has been especially dwelt upon by Bouillaud. Its influence in affecting the circulation in the brain is undeniable, but it is probable that the frequency with which it induces hæmorrhage, either in the lungs or in the brain, has been over-rated. In most, if not all, of the cases on record of cerebral apoplexy connected with cardiac hypertrophy, the result was more justly attributable to the coincident arterial disease than to the increased impulse of an enlarged heart. Pulmonary apoplexy appears rather to be connected with the obstructions to the circulation presented by mitral disease, and with the blocking up of branches of the pulmonary artery, than with an hypertrophic condition of the heart. With regard to the liver, we find that in fatty degeneration of the heart it commonly presents a similar concomitant affection, not to speak of the congestion to which it is almost invariably subject when the return of the blood to the heart is in any way impeded. Congestions of the venous system of the entire body are frequent in cardiac disease, and manifest themselves by lividity of the cutaneous surface, and of the mucous membranes; and the secondary effects of stasis are shown in these tissues by œdema and hæmorrhage, while in the serous cavities they are evidenced by an effusion of serum—one form of passive dropsy. Of the latter we find the peritoneum chiefly prone to suffer, a circumstance which we may fairly attribute to the absence of any compression, such as we find normally exerted upon all the other serous sacs.

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* Cayley: "Trans. Path. Soc.," vol. xvii. 1866, p. 86.

† "Trans. Path. Soc.," vol. xvi. 1865, p. 91.

‡ "Medico-Chirurgical Trans.," vol. xxviii. p. 536.

CHAPTER XXII.

CONGENITAL MALFORMATION OF THE HEART.

CARDIAC anomalies, dependent on congenital malformation, are mostly due to what is termed arrest of development; that is to say, the organ does not go through those changes by which it is transformed from its original simple structure into the complicated organization necessary for the performance of its functions. Hence such anomalies are characterized by greater simplicity; an excess of development is rarely met with in the heart, except as an acquired condition. In all these varieties of congenital arrest, we observe a tendency to return to the primitive type of a single pulsating cavity. As the growth proceeds through its different stages, from the simplest condition of the pulsating vessel, to the complex mechanism of the perfect heart, we see close resemblances between temporary conditions of the human heart, and permanent conditions of the heart in the lower animals. Thus the type of the piscine heart is presented in those cases where in a man we only find a single auricle and ventricle. There are, however, very many instances of congenital malformation which may be referred to disease (inflammation) occurring during intra-uterine life.

Classification of Malformations.—Such anomalies are conveniently classified, according to the period of fœtal life at which arrest of development takes place. Thus, according to Dr. Peacock* (from whom we have borrowed much of the subsequent summary) there may be (1) anomalies dependent on arrest of development at an early period of fœtal life, or probably from the fourth to the sixth week, so that the organ retains its most rudimentary form; the auricular and ventricular cavities being still single, or presenting very slight indications of division, and the primitive arterial trunk being retained, or the aorta and pulmonary artery very imperfectly evolved. (2) Those in which the arrest occurs at a more advanced period, probably from about the sixth to the twelfth week, when the auricular and ventricular partitions have been already partly formed, and the aorta and pulmonary artery more or less completely developed. Such are the cases in which, with imperfect separation of the ventricles, the arterial or auriculo-ventricular apertures are constricted or obliterated, and the origins of the primary vessels, and especially of the aorta, are misplaced. (3) Cases in which development has gone on till a later period, so that the auricular and ventricular septa are completely formed, and the primary vessels possess their natural connections; but in which disease either prevents the natural evolution of the organ after birth, or produces conditions which are the cause of more serious obstruction in after life.

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* "Malformations of the Heart," 2nd edition, 1866.

I.—MALFORMATIONS DEPENDENT ON EARLY ARREST OF DEVELOPMENT.

In these cases the heart may, it is said, consist of but a single contractile cavity, but this has been observed only in imperfectly formed fœtuses. In infants which have survived for some days after birth, a heart of two chambers has occasionally been found. The common ventricle usually gives origin to a distinct aorta and pulmonary artery, but sometimes the former supplies the lungs by a ductus arteriosus. The common auricle receives the venæ cavæ and pulmonary veins, but usually shows some indication of a division; and is sometimes provided with one or two auricular appendages. In cases where development has advanced somewhat farther, three chambers are found, viz., two more or less distinct auricles, and one ventricle. The two auricles receive the pulmonary and systemic veins respectively, while the single ventricle either gives origin to a distinct aorta and pulmonary artery, or else the latter arises from the former by a ductus arteriosus. There is sometimes a rudimentary ventricular septum. This kind of malformation, though very rare, is of more frequent occurrence than the biloculate. It has been observed in individuals who have lived to the age of 11, 16, or even 24 years, though more generally in infants who have survived their birth only a few days.

II.—MALFORMATIONS DEPENDENT ON ARREST OF DEVELOPMENT AT A LATER PERIOD.

In specimens classed under this head all four cavities are present, but there is some imperfection in one or both of the septa, and usually some obstruction either in the aorta or pulmonary artery. Imperfection of the inter-ventricular septum, if only partial, usually occurs at the base, where, during fœtal life, the division of the cavities is last effected, and here more especially in a triangular area, where the ventricular septum is on the left side uncovered by muscular substance, and which is known as the "undefended space." There may, however, be deficiencies in other parts of the ventricular septum. These defects may coexist with imperfect separation of the auricles, but such is, according to Dr.

Peacock, not always the case; and on the other hand, the auricular septum may be imperfect, or the foramen ovale unclosed, while the partition of the ventricles is entire. Openings at the part called the undefended space are thought to be sometimes caused by disease, and not congenital. Deficiencies in the partitions of the auricles and ventricles usually coincide with other important anomalies, especially with some source of obstruction at the pulmonic or other orifice, so that we may often regard the former as the consequence of the latter; but this does not always hold, large apertures being sometimes found in the septa without other abnormalities of any kind. In these cases there have usually been no signs of obstruction or disorder of the circulatory system during life.

Deficiencies in the auricular septum are also found unaccompanied by any other anomaly. This defect is sometimes coincident with imperfection of the foramen ovale and its valve, but sometimes found at another part of the cavity.

Defects in the Septa, associated with Constriction of the Orifices, or Misplacement of the Primary Vessels.—One of the commonest varieties of malformation of the heart consists in the association of these two forms of anomaly. Obstruction to the flow of blood from the right ventricle is the condition most frequently met with. This obstruction may occur, according to Dr. Peacock, either (1) at the free edges of the valves of the pulmonary artery; (2) at the line of attachment of the valves to the fibrous zone; (3) in the course of the pulmonary artery itself; or (4) at the commencement of the infundibular portion of the right ventricle. There may also be a defect which can only be described as congenital smallness of the pulmonary artery. In all these cases we meet with the same associated defects in other parts of the heart; viz., imperfection of the inter-ventricular septum, and usually some deviation of it towards the left, so that the right ventricle is of large size, and gives origin, wholly or in part, to the aorta. The foramen ovale sometimes remains open in such cases, and sometimes not. The ductus arteriosus is sometimes pervious, but in other cases is prematurely closed. The latter anomalies are probably to be regarded, as first pointed out by William Hunter, as consequences of the obstruction to the flow of blood from the ventricle; the excess of pressure thus caused preventing the normal separation of the cavities. Premature closure of the ductus arteriosus has, however, been regarded as a cause of narrowing of the pulmonary artery. The amount of obstruction in the pulmonary artery may vary from total occlusion (a condition generally incompatible with life for more than a few days) to mere narrowing of its calibre, which may give rise to few or ambiguous symptoms during life, and be unaccompanied by any other important anomaly, except hypertrophy and dilatation of the right ventricle. The latter changes are (as might be expected) most marked in persons who survive for some years. There is usually more or less cyanosis, but this is not invariably the case; shortness of breath, and a tendency to syncope being sometimes all that are observed. A systolic murmur may generally be heard on the left side of the sternum, especially over the third costal cartilage, and there may be at the same spot a palpable thrill. It has been pointed out by Lebert, that in many cases of narrowing of the pulmonary artery, tubercular phthisis occurs in the lungs.*

Origin of both Aorta and Pulmonary Artery from Left Ventricle.—This anomaly may be theoretically accounted for by supposing the ventricular septum to be transposed towards the right, being at the same time imperfect, just as, when both great vessels are connected with the right ventricle, the septum appears to deviate towards the left. It is, however, a very rare form of anomaly.

Obliteration or Obstruction of the Aorta, with or without Imperfection of the Ventricular Septum.—This defect is singularly rare, as compared with the corresponding defect of the right side of the heart. When it occurs the pulmonary artery supplies the aorta with blood through the ductus arteriosus. The right ventricle is very large; the left may, if the septum be perfect, be almost obliterated. Or it may, if imperfectly separated from the right ventricle, assist in the origin of the pulmonary artery. In the former case the foramen ovale must be widely open, and the blood from the lungs thus reaches the right auricle and ventricle.

Transposition of the Origin of the Great Vessels.—It sometimes happens that the pulmonary artery is found originating from the left ventricle; the aorta from the right. This condition has been observed in hearts presenting no other serious defect, except patency of the ductus arteriosus and foramen ovale. The auricles receive blood as usual, the right from the systemic circulation; the left from the lungs. The result accordingly is that, except for the small communication effected by the passages just mentioned, there are essentially two hearts; or (in the words of Baillie †) there must be florid blood circulating between the lungs and the left side of the heart; a dark-coloured blood circulating between the general mass of the body and the right side. In one instance of a child which survived two years and eight months, the ductus arteriosus was closed but the foramen ovale widely open. The origin of this curious anomaly is involved in great obscurity, and no adequate explanation has yet been suggested.‡

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* Cases of constriction or obliteration of the pulmonary artery or orifice of the right ventricle are recorded in the "Transactions of the Pathological Society," vol. i. pp. 50, 52, 58, 204, 205; vol. ii. p. 37; vol. iv. p. 81.; vol. v. pp. 67, 99; vol. vii. p. 89; vol. viii. pp. 107, 123, 167; vol. x. pp. 89, 90; vol. xi. p. 45; vol. xiii. p. 42, 57; vol. xv. 60, 89; vol. xvii. 45. Many other references are given by Dr. Peacock in the work so often referred to, "Malformations of the Heart," 2nd edition, 1866.

† Engravings to illustrate "Morbid Anatomy," p. 23, plate vi. fasc. 1. 2nd edition, London, 1812.

‡ Cockle: "Med.-Chir. Trans.," vol. xlvi. p. 204, 1863. Ward: "Path. Trans.," vol. iii. p. 63, 1851. Kelly: "Path. Trans.," vol. xxii. p. 92, 1871. See also Dr. Peacock's work.

III.—MALFORMATIONS DEPENDING UPON PREMATURE CLOSURE, OR PERMANENT PATENCY OF THE FŒTAL OPENINGS.

The changes which are rendered necessary in the fœtus after birth by the cessation of the placental and the establishment of a pulmonary circulation, affect chiefly two parts, namely, the ductus arteriosus and the foramen ovale; and these openings may be either prematurely closed or remain unduly patent.

Premature closure of the ductus arteriosus is of course an anomaly belonging to fœtal life, but the results first become apparent after birth. The consequence of it is that the pulmonary artery gives passage during fœtal life only to the very small amount of blood which circulates through the lungs, and is in consequence so imperfectly developed as to be incapable of adequately expanding after birth and of conveying the larger quantity of blood which should then be transmitted. The small size of the vessel thus becomes a source of permanent obstruction, which entails other defects in the development of the organ. (Peacock.)

Premature closure of the foramen ovale leads to excessive development of the right side of the heart and corresponding imperfection of the left side. A case of this description is recorded by Mr. Pye Smith in the first volume of the "Pathological Transactions," where this opening was entirely wanting in a child which survived its birth only twenty-one hours.

Permanent Patency of the Ductus Arteriosus.—This condition may occur in combination with other important anomalies; especially closure or obstruction of the first portion of the aorta (ascending portion of the arch) and the same defect in the commencement of the pulmonary artery. In the former case the open duct serves to convey blood to the descending aorta and even to the arch; while in the latter case it is the channel by which blood reaches the lungs. In other cases, however, it may be the only or chief anomaly present. It has been supposed that in such cases it is a consequence of previous obstruction in the pulmonic circulation.

Permanent Patency of the Foramen Ovale.—This defect may be found in different degrees, and under various circumstances. The opening may be either unusually large or of the natural size, and the valve either the normal size or defective. Defect of the valve may amount to total absence or may be simply inadequate development; or, finally, there may be larger or smaller perforations in it. Such anomalies as these are usually found associated with other serious defects of the heart, and may often be ascribed to undue pressure in one or other auricle during the development of the organ, from obstruction of some of the great vessels. The opening may then have a diameter of half an inch to an inch, and in such cases there is always evidence of some serious defect in the heart. It often happens, however, that an open foramen ovale is found after death in persons who have presented no symptoms whatever of disordered circulation during life.

The commonest form of imperfection is when the membranous curtain covers the opening entirely, but there is a "valvular" opening through which a probe can be obliquely passed. This does not, in all probability, at all interfere with the functions of the heart, so long as the blood pressure remains (which it must be normally) equal on both sides. If, however, any cause, such as obstruction in the lungs, produce a higher pressure on one side of the septum, blood may pass from one auricle to the other. By this inequality of pressure, combined with the consequent dilatation of the auricle, the exent of the opening will be greatly increased.

The form of defect which we believe to be of practical importance, and to be in all probability never repaired, is when the curtain leaves a crescent-shaped opening at its lower border, being attached only by two pillars at the corners; and, further, has two or three perforations arranged in a curved line above. The curtain is at the same time usually very thin. This form of valve we have seen both in infants dying soon after birth with obviously imperfect circulation, and in adults who have suffered from dyspnœa and some degree of cyanosis.

A single crescent-shaped opening below, with the curtain well formed, is possibly the result of dilatation of the auricle in after life.

MALFORMATIONS OF THE VALVES.

The valves frequently present a congenital arrest, or excess of development, sometimes independent of any other malformation of the heart, but commonly associated with further lesions. In reference to the arterial orifices, Dr. Peacock * oberves, that the aperture may be defended by a single valve, protruded forwards in the course of the circulation, a condition seen chiefly in the pulmonic artery, or only two valves appear, owing to two having united at their edges; or again, there may be two fully developed semilunar valves, with an abortive valve intervening. Of forty-one cases of defect in the number of the valves, Dr. Peacock found the malformation at the pulmonic in nine; in thirty-two, at the aortic orifice. Of fifty cases of malformation of the semilunar valves examined by the same observer, nine were examples of excessive development. Of these he found that eight were cases in which the pulmonic valves were in excess, and in one only was there more than the natural number of valves at the aortic orifice.

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* "Report of the Pathol. Society," 1852, p. 292.

"In some cases the excess in the number of valves seems to be due to the division of one of them into two, such divided valves being smaller in size than the others. In others there are three valves of nearly equal size, with a smaller supplementary valve interposed between two of them. Occasionally the aperture is provided with four valves, gradually decreasing in size, and in the other cases there may be four valves of nearly equal size, and natural form." Similar defects are met with in the tricuspid and mitral valves, but more rarely. We sometimes find the mitral, or less frequently, the tricuspid valve with all the flaps adherent by their edges, so as to form a funnel-shaped structure. This peculiarity is probably always congenital, but may be confounded with inflammatory conditions.

Presence of Muscular Fibre in the Valves.—This peculiarity, probably congenital, though not strictly a malformation and not a disease, may be mentioned here. The mitral and tricuspid valves are sometimes found to contain muscular fibres which are continuous with true muscular cords normally found in place of some of the tendinous cords by which the valves are attached. In one case observed by Dr. J. Ogle * a patch of muscle as large as a fourpenny piece was seen in the anterior flap of the mitral valve. The fact seems to be that, as first shown by Kürschner, minute bundles of muscle are very frequently, if not normally, present in the valves, and are occasionally hypertrophied so as to form recognizable masses. The true explanation seems to have been suggested by Savory ("Philos. Mag.," March, 1852,) that the muscular attachments of the valve overlap in a greater or less degree its free border.

CYANOSIS.

Cyanosis is a term applied to a livid, purplish hue of the cutaneous surface, which is found to accompany some organic and congenital disturbances in the central organ of the circulation, which have just been enumerated. It is of a more intense character than the slaty tinge which the complexion is very frequently observed to assume in acquired disease of the heart. It was formerly attributed, on theoretical grounds, solely to one lesion, a permanent patency of the foramen ovale; and although this occasionally gives rise to the affection, by allowing an intermixture between the blood of the two sides of the organ, and causing the mixed fluids to be circulated through the system, without having undergone the purifying process to which the blood ought to be subjected in the lungs, it is satisfactorily demonstrated, both that the foramen ovale may remain open, to a considerable degree, throughout life, without inducing any serious disturbance of the circulation; and on the other hand, that various other irregularities in the heart, such as those before referred to, may give rise to cyanosis. Bizot found the foramen ovale more or less open in forty-four out of one hundred and fifty-five subjects, in none of whom was there a trace of the morbus cœruleus. Two openings have been found in the ventricular septum, and no cyanosis resulted; a marked instance of this kind in an individual who attained the age of eight years, was brought before the Pathological Society, by Dr. Quain, in 1847. In such a case we are justified in assuming that the forces of the two sides of the heart are so exactly balanced as not to disturb the circulation; and the orifice of the pulmonary and systemic arteries being patent, the contents of each side pass into their proper channel. That this is a prevailing law for many cases of cyanosis, is shown by the fact, that it frequently does not manifest itself unless there is some further cause for derangement of the circulation, such as bronchitic affections, to which, it may be remarked, cyanotic individuals are peculiarly subject.

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* "Trans. Path. Soc.," 1858, vol. ix. p. 109, plate v.

Another lesion that appears to be often associated with cyanosis is a contracted state of the pulmonary artery; and, as in that case more than usual pressure will continue to be exerted upon the foramen ovale, this will necessarily remain patulous, and allow a passage of blood from the right to the left auricle; in such a case it may be almost looked upon as a safety valve. Gintrac* has analyzed fifty cases of cyanosis, and among them found obstruction at the pulmonic orifice in twenty-six; the proportion is stated to be still greater by other authors. But the blue disease is not necessarily the result of an admixture of the contents of the two sides of the heart; anything causing an arrest in the return of the venous blood to the heart is sufficient to give rise to it. In a case of marked cyanosis, recorded by Mr. Ebenezer Pye Smith, already referred to, in which the foramen ovale was perfectly closed, and had evidently been so for some time before birth, there was no interventricular communication, the left ventricle was almost obliterated, and the aortic opening was also very small, being about two lines wide. Here, then, there was an evident arrest at the aortic orifice, which reacted upon the pulmonary circulation, and through that upon the systemic capillaries. The lungs were too much charged with blood to perform the duty of aëration effectually, and a congested or cyanotic condition of the surface resulted. Other similar instances of the cyanosis being due to contraction at the aortic orifices are on record.

One of the most palpable instances that has occurred to us, proving how little we are able to account for cyanosis theoretically, was that of a child that lived to the age of nine weeks, and whose heart, after death, was found to present no auriculo-ventricular opening, on the right side, while there was scarcely any inter-ventricular septum at all. Here there had been no cyanosis, although a thorough intermixture of the venous and arterial blood must have necessarily taken place.

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* "Sur la Cyanose," Paris, 1824.

Bouillaud* is of opinion that the communication between the two sides of the heart, and the consequent admixture of the arterial and venous blood has, comparatively, little to do with the purple hue of the complexion, which he considers to result, mainly, from the coincident obstacle offered to the circulation by a malformation of the arterial orifices of the heart. The numerous cases on record in which not only the foramen ovale was patulous, but in which there was further evidence of the actual passage of the blood, directly from one side of the heart to the other, shows, as Dr. Peacock † has remarked, that there is a want of just relation between the amount of venous blood entering the general circulation and the degree of cyanosis. The lesions that are found in connection with this symptom, consequently, require to be carefully analyzed before we can determine the exact part that each bears in its production. They may shortly be enumerated as a patulous condition of the foramen ovale, from the valve not entirely covering the orifice (with which a defective involution of the Eustachian valve is commonly combined) permanent patency of the ductus arteriosus; contraction of the systemic or pulmonary arterial orifices; a deficiency in the inter-ventricular septum; and the malformation in which the aorta springs from both ventricles.

History and Symptoms.—Cyanosis is a disease which generally shows itself at or immediately after birth. The circumstance that it occasionally makes its appearance later in life has induced Meckel and Abernethy to assume that the foramen ovale may re-open, an hypothesis which is unnecessary, as we now know how frequently a communication exists between the auricles, without producing cyanosis, and that this lesion may, under certain circumstances, as in diseased states of the lungs, induce a disturbance in the balance of the circulation, sufficient to force the blood through the auricular septum.

Stress has been laid by several authors upon the circumstance that the fingers of cyanotic individuals are found clubbed. We only advert to it to mention that it is by no means diagnostic of this form of heart-disease, or, in fact, of any distinct malady. A more important point is an observation with which Rokitansky concludes his remarks on the subject, to the effect that cyanosis is incompatible with tuberculosis, against which he states that it offers a complete protection. We do not deny that this is the prevailing rule, yet it is not as absolute as the author quoted asserts. In the Report of the Pathological Society for 1848 (p. 200) we find a case presented by Dr. Peacock, which refutes the universality of the law. There the post-mortem examination of the individual, a young man, aged twenty, established the following facts:—The right lung was extensively permeated by tubercle, and towards the apex exhibited several small cavities; the left lung contained much solid tubercle; the heart was hypertrophic; the pulmonary artery exhibited a complete diaphragm, formed by adhesion of the valves, leaving only a small triangular aperture; the foramen ovale was very widely patulous. There had been cyanosis during life, but not in a very marked degree.

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* "Traité Clinique," &c., vol. ii 690, et seq.

† "Pathol. Reports," 1848, p. 202.

CHAPTER XXIII.

THE BLOOD VESSELS.

THE arteries and veins are not by any means subject to precisely the same morbid conditions; the difference in the direction of the current, in the composition of the blood, in the velocity and force of the circulation, and in the structure of their coats, are points that must not be overlooked in forming an estimate of the diseases of these two great classes of vessels. The manifestations of disease in its primary and secondary form are essentially different in the two, as we find their physiological and anatomical relations to be widely apart. The arteries exhibit between their lining membrane and cellular coat a dense fibrous layer, which contains no vessels, and therefore removes the vasa vasorum, which ramify in the cellular coat, much further from the lining membrane than is the case in the veins, nor can any vessels be detected on the lining coat, or between it and the middle tunic.

The bearing of these anatomical facts on the diseases of these two classes of vessels respectively will be seen as we go on.

THE ARTERIES.

To proceed systematically, we shall first examine the morbid conditions of the arteries.

We have seen that it is a subject of debate whether the middle and lining coats of the arteries are subject to inflammation; as they possess no blood-vessels of their own, we can scarcely assume them to present symptoms of the primary phenomena of inflammation; but that they may be secondarily involved in inflammatory affections proceeding from their outer coat cannot be doubted. Acute inflammation of the outer coat, or periarteritis, has been very carefully studied by Virchow, and it is the only form of acute inflammation of arteries of which the existence can be said to be clearly established. The cases formerly described as acute arteritis, in which sudden obliteration of a vessel was accompanied by acute inflammation are now looked upon with suspicion; since we cannot be sure that the blocking of the artery was not the primary occurrence and cause of the other symptoms. The whole train of phenomena precisely corresponds with what is now called embolism. Of late years such cases have rarely been described, and excellent observers, with unusual opportunities for observation, as Dr. Wilks, have never seen a single case. So that the older observations must now be accepted with some degree of caution.

Bizot describes as the result of acute inflammation of the arteries, an "albuminous exudation" of greater or less thickness, of the consistency of jelly, transparent, smooth, sometimes rose-coloured, at others colourless, covering the lining membrane. It is occasionally so transparent as to escape attention unless very carefully examined. It occurs in patches, solitary or numerous, and diminishes the calibre of the vessel; in one case Bizot saw it entirely plugging up the anterior tibial artery. In the aorta this exudation is formed mostly at the orifice of the arteries arising from the arch, at the mouth of the cœliac, mesenteric and renal arteries, and at its posterior surface, so as to block up the mouths of the intercostal arteries.

More recently MM. Cornil and Ranvier* have described acute endarteritis in somewhat similar terms, as characterized by swelling of the internal coat in the form of flat patches of various size, in colour and consistency resembling the "exudations" described by Bizot. These plates are found to be in great part composed of round embryonic cells, which result from the proliferation of normal elements of the internal coat, which is most active near the surface. They insist upon the fact that this multiplication of elements on the surface of the internal coat is peculiar to acute endarteritis, and distinguishes it from chronic atheromatous endarteritis (which will be presently spoken of), where the same changes go on in the deep layer of the internal coat. Cornil and Ranvier have on several occasions seen this process in the aorta; they regard it as rare in medium-sized and small arteries, but common in the vessels contained in the granulating tissues of wounds. Though these views are opposed to those which have been prevalent of late years, when it has been customary to deny the existence of acute endarteritis, there seems no reason why this should not occur here as well as in the corresponding tissue of the endocardium and in the internal coat of veins; where we have reason to believe such a process does take place.

As an instance of what was formerly called arteritis, we may take the following case of "acute inflammation of the aorta," recorded by Mr. Hodgson.† A man was seized with violent pneumonia, which proved fatal in five days. The cadaveric inspection exhibited "all the thoracic viscera in the highest degree of acute inflammation; the aorta was also involved, its internal coat being of a deep red colour, and a considerable portion of lymph being effused into the cavity. The effused lymph was very intimately connected with the internal coat of the vessel, and a plug of it had extended into the left subclavian artery, and nearly obliterated the cavity of that vessel." Another important case is very fully described by Romberg.* In reference to this subject some experiments performed by Gendrin† were formerly thought to be of considerable importance in demonstrating the capability of the coats of the artery giving rise to inflammatory exudation in the strict sense of the word. He found that on injecting an irritant substance into a portion of an artery included between two ligatures, and deprived of blood, a deposit of coagulable lymph took place, which covered the internal coat, and at last formed a plug filling up the channel. The lining membrane at first was only slightly discoloured, and through it a network of injected capillaries might be distinguished on the adherent surface of this tunic to the middle coat. When the inflammation had advanced this was no longer seen, the internal coat having become pulpy, rugous, and dull. The suppuration that followed did always coincide with ulceration of the inner coat; the pus, however, was not necessarily deposited in the vessel, but infiltrated into the cellular sheath, forming small abscesses. Similar experiments were made by Rigot and Trousseau with negative results; and by others with various consequences.

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* "Histologie Pathologique," 1873, deuxième partie, p. 530.
† "On the Arteries," p. 5.

Traumatic and Secondary Arteritis.—In the more common form of inflammation of arteries produced by laceration, pressure, ligature, or the inflammation of neighbouring parts, the outer and middle coats are chiefly or solely affected, and it is hence called by some periarteritis and mesarteritis. The outer coat is found brightly injected, and with the middle coat swollen and infiltrated with exudation, while the inner coat is thrown into folds, and ultimately becomes necrotic, and is detached. Diffuse suppuration may result in the substance of the arterial walls. The blood contained in the vessel will ultimately coagulate, and thus produce all the consequences of arrest of circulation in the part which the vessel supplies.

Results of Arteritis.—From the time of J. P. Frank,‡ who first drew attention to the subject of arterial inflammation, to the most recent periods, various pathological conditions have been attributed to it; the acute forms have been repeatedly asserted to be the cause of trismus neonatorum, a disease which at present is one of very rare occurrence among ourselves. Dr. West denies this cause, but Dr. Collis,§ and recently Dr. Schöller‖ satisfied themselves of its real existence. The latter found inflammation of the umbilical arteries in fifteen out of eighteen cases of trismus neonatorum. There was tumefaction of the umbilicus, reddening and congestion on the external surface; the channel contained pus, and the lining membrane was eroded and invested with an albuminous exudation. Dr. Schöller has carefully examined these parts in all other new-born children who died shortly after birth, and has never succeeded in discovering similar lesions. It does not appear that traumatic tetanus in the adult, to which we may compare trismus neonatorum, is accompanied by similar lesions.

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* "Manual of Nervous Diseases," Sydenham Society's edition, vol. ii. p. 238.

† "Histoire Anatomique des Inflammations," vol. ii. p. 13.

‡ "De curandis Hominum Morbis," vol. ii. p. 363.

§ "Dublin Hospital Reports," vol. i. p. 285.

‖ "Neue Zeitschrift für Geburtskunde, herausgegeben von Busch, d'Outrepont und Ritgen," vol. v. p. 477.

CHRONIC ARTERITIS, OR ATHEROMATOUS DISEASE.

With regard to chronic arteritis the opinions of writers are as much divided as in reference to the acute form. The same difficulties in determining the relation of cause or effect in this subject have been felt by most of the writers on the subject, and have not yet met their complete solution. The older authors attributed the appearances of chronic arteritis to syphilitic taint or mercurial poisoning; some of the more recent, among whom we may mention Corvisart,* have held a similar opinion, and Hodgson† supports it on the ground that he has observed aneurism and those organic alterations which generally attend the formation of aneurism, to prevail in subjects that have suffered from venereal disease, and who have taken large quantities of mercury. The connection of this disease with syphilis is moreover supported by many authorities in the present day, especially by the medical officers in the army, who have unusual opportunities of observation in venereal disease.‡

The majority of authors are now of opinion that fibrinous deposit, atheroma, ulceration, ossification, and aneurism are the result of a chronic inflammatory process, though there are some who still regard it as essentially a degenerative process.§ We understand by it a chronic inflammatory process, starting in the inner coat and at first hyperplastic, but usually resulting in degeneration, either of the fatty or calcareous kind. The former is the most marked feature of the disease, and has here some peculiarities which have obtained for it a special name, *atheroma*, and the disease is hence often called the atheromatous process.

Three stages may be distinguished in chronic arteritis.

The first stage does not often come under our cognizance, and when it is found later stages usually exist simultaneously in other parts. At this period of the disease the inner coat of the artery appears irregularly thickened or elevated by masses of a greyish translucent material, looking as if deposited upon its surface. Careful examination, however, shows that the seemingly adventitious material is really continuous with and formed from the inner coat of the artery. The innermost lining, or endothelium, may generally be traced uninterruptedly, but the thickening affects the intima proper, and not the middle coat. The material is, when newly formed, greyish, translucent, and sometimes like cartilage in appearance, in general resembling the substance of the translucent nodules formed on the valves of the heart in acute endocarditis.

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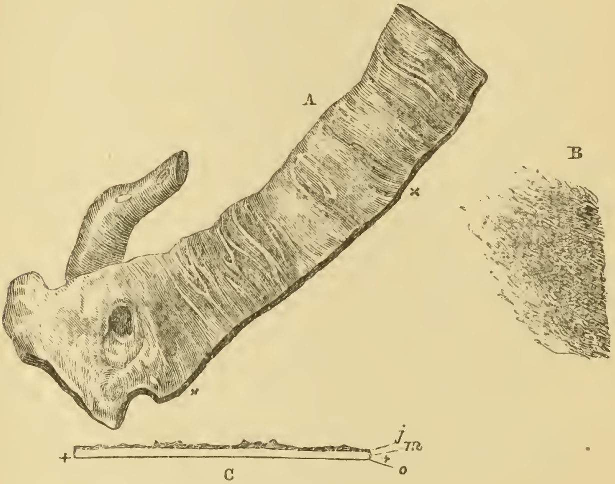
* "Essai sur les Maladies du Cœur," p. 319.

† "On the Arteries," p. 9.

‡ "Reports of the Army Medical Department," vol. v. p. 481, and vol. xii. p. 381.

§ Rokitansky formerly regarded the process as consisting essentially in the *deposit* of a morbid substance from the blood current upon the inner coat, this substance being itself regarded as identical with the fibrin of inflammatory exudation. In the later editions of his work he has, however, modified, if not abandoned, this view, and the *deposit* theory, which rested chiefly on his authority, is now generally given up.

Fig. 89.



Incipient atheroma and fatty degeneration of an iliac taken from an aged female. The lining membrane is much puckered, owing to the irregular thickening between it and the middle coat.

A. Naked-eye view of the artery; B. microscopic appearance of the fibrinous material, dotted with oil molecules; C. a longitudinal section of the artery taken between *—*; *j.* the inner coat much thickened; *m.* middle coat unaltered; *o.* the external coat.

This translucency and grey colour of the material are soon lost, and it becomes yellow and opaque. This change, which is owing to the deposition of fatty molecules in the substance, marks the commencement of the *second stage.* It is the condition which we most commonly recognize as atheromatous degeneration of the arteries, but where it occurs, evidences of more advanced forms of degeneration are also invariably present. These subsequent changes assume one of two forms: either fatty degeneration (atheromatous degeneration properly so called), or calcareous metamorphosis.

Advanced fatty degeneration, of which the earlier stage, as we have seen, produces yellowness and opacity in the new-formed mass, causes its softening and liquefaction. The mass, which was previously tough and resistent, becomes soft and crumbling, and finally breaks down into a sort of yellow pasty mass properly called atheroma,* or an "atheromatous abscess."

This change first takes place in the deep layer of the inner coat, bordering on the middle coat, and thus the softened mass or abscess appears to be covered by the whole thickness of the inner coat. It is this which has led to the opinion that the whole atheromatous process takes place beneath the inner coat, or between it and the middle coat. The process of degeneration, however, gradually spreads inwards and outwards. Inwards it involves the whole thickness of the inner coat, till the softened mass is covered only by a thin membrane. Outwards the same degeneration may extend into the middle coat, the connective tissue and muscular fibres of which may both be affected.

When the thinning of the inner coat becomes extreme it is absorbed or ruptured, forming either a large opening or else minute perforations. If the former, an excavation like an ulcer may result. By these openings doubtless some "atheromatous" matter must escape into the blood, but the conditions or consequences of this have not been traced. On the other hand, blood enters the depression or cavity, and becoming coagulated, the degenerated blood-clot is mixed with the atheromatous matter. Small masses of coagulum, or "vegetations," may also be formed on the edge of such an excavation.

Calcareous Degeneration.—The second form of advanced degeneration is the calcareous, which has also been termed "ossification" of arteries. It must, however, be remembered that (as in the case of fatty degeneration) calcification of some part of the coats of the artery may occur quite independently of the chronic inflammation connected with atheromatous disease. When it occurs as a result of the disease now under consideration, the process consists in the deposition of calcareous matter in the thickened inner coat in such quantity as to convert this into a bone-like mass. The innermost layer is at first unaffected, and passes smoothly over the calcified structures, but after a time this is either calcified or absorbed, so that the calcareous plates lie bare to the blood-current. In the other direction calcification may extend into the middle coat till a considerable thickness of the whole arterial wall is thus affected, and plates of even a line or more in thickness may result. The calcareous masses thus produced project in the most varied and fantastic forms, into the current of the blood; thus, in its turn, giving rise to further deposits in the shape of fibrinous coagula; or the bony deposit advances under the lining membrane, till it encircles the artery and converts it into a rigid channel, inducing that condition which is found to accompany senile gangrene, fatty degeneration of the heart, cerebral softening, and other morbid processes.

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* Etymology, from ἀθήρη=groats or meal; a pulp made thereof; hence, ἀθήρωμα =a tumour filled with similar substance (Galen).

Calcification of the Middle Coat.—The process of calcification is often complicated by a similar affection of the middle coat, which is met with chiefly in the smaller or medium-sized arteries, where the muscular middle coat predominates, and sometimes occurs alone. The calcareous matter is here deposited in the muscular bundles themselves, which become rigid, opaque, and yellow. The change is, however, partial, and the remaining part of the muscular coat shows hypertrophy. This change is obvious from outside, even to the naked eye, by the transverse and annular stripes which correspond to the seat of calcification, plainly showing that this is in the transverse fibres of the middle coat. The same thing is well seen on making a longitudinal section in the vessel, when the cut ends of the calcified muscular fibres are seen in the midst of the fleshy middle coat, which is evidently abnormally thick. The hypertrophy and subsequent degeneration are probably part of a chronic process depending on excessive arterial tension. It is extremely local and partial in its distribution.

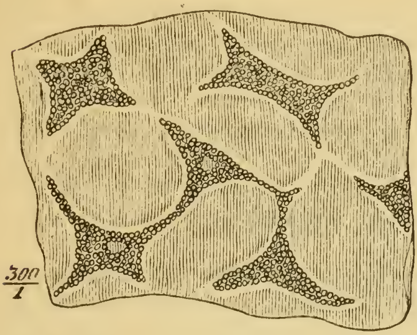
Consequences of Calcification.—Calcification of the arteries affects the circulation in two ways. In the first place, the smaller arteries are actually diminished in their calibre by the calcareous deposit, and the passage of blood is thus hardened. In the second place, the general loss of elasticity caused by the rigidity of the walls is an actual obstruction to the circulation, more force being required to propel fluid through a rigid than through an elastic tube. Hence there will be positive retardation and enfeeblement of the blood current, unless compensatory hypertrophy of the heart be produced, which restores the balance.

Calcification without Atheromatous Degeneration.—This condition, though rare, occurs in some well-marked instances. It usually affects all the coats of the vessel, which is thus converted into a calcareous shell. This affection has been represented as one of the consequences of "calcareous metastasis," or the removal of lime from one part of the body and its deposit in another part. In other cases it appears to be the result of acute inflammation. In any case it would seem to be a comparatively rapid, not a chronic process.

Fatty Degeneration, independent of the Atheromatous Process.—The coats of arteries, like many other parts of the body, are subject to simple fatty metamorphosis. This affects the inner coat, producing yellowness and opacity, generally partial, so as to give a cloudy appearance to the whole. The minute changes consist in fatty degeneration of the stellate, or angular connective tissue corpuscles, and ultimately of the intercellular substance. (See Fig. 90.)

The same process in the middle coat produces fatty degeneration of the muscular fibres, which is not uncommon. It may also affect the outer coat, and in this part is very common in the smaller arteries, where it usually accompanies a similar change in the capillaries. In the arteries of the brain this change is seldom wanting, and is very conspicuous among the evidences of old age; still there is a peculiar form of fatty degeneration of the external coat, which is specially prevalent in children. Precisely the same changes may be traced in the small arteries and capillaries of other organs, such as the kidneys and lungs — though the vessels are of course not so easily isolated.

FIG. 90.



Connective tissue corpuscles from the inner coat of an artery, in a state of fatty degeneration. (After Rindfleisch.)

Coagulation in Arteries.—The formation of a coagulum in the artery is a well-known physiological effect of the laceration by mechanical or other means of the lining membrane, and the atrophy or gangrene of the part nourished by the artery is an illustration of the effects following similar obliteration of the channel from disease. We have alluded to the cerebral affections resulting from an arrest in the arterial circulation; senile gangrene is another morbid condition which has been ascribed, by Dupuytren and Cruveilhier, to arteritis; or, as would now be said, to obstruction of the diseased artery by formation of coagula. It is now doubted whether arteritis ever gives rise to suppuration, but independently of the cases of suppuration in the umbilical artery quoted from Dr. Schöller, Andral's * and Hodgson's authority, † were formerly thought to determine the question ; for these authors state that actual idiopathic suppuration does occur in the artery.

The spontaneous coagulation of the blood in the arteries is not, however, usually the result of inflammatory action. It may occur in consequence of a peculiar condition, which favours the chemical tendencies, that normally ought not to come into play until after death. This spontaneous coagulation is especially met with in the pulmonary arteries, where the occurrence of the inflammatory symptoms has, as yet, not been met with. Sir. J. Paget,‡ in describing a case of the kind, says, that nearly all the branches beyond the primary divisions of the pulmonary artery contained clots of blood, which from a comparison with those found in tied arteries he judged to be from three to ten days old. The clots did not commonly extend continuously from any large branch of the pulmonary artery into many of its successively subordinate divisions, no branch of the pulmonary artery less than half a line in diameter appeared to contain any of these clots, and the pulmonary veins were healthy and empty. The case under consideration proves that a large portion of the pulmonary circulation may be arrested for a considerable period without immediate danger to life, a circumstance explained by Sir J. Paget by assuming a retardation of the circulation in the systemic vessels, in order to allow the quantity traversing them in a given time to be equal to the reduced quantity which in the same time traverses the lungs. In order to keep up the necessary balance, the systemic circulation is as much less rapid than the remaining pulmonary circulation is more rapid, than before the obstruction took place.

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* "Anat. Pathologique," tom. ii. p. 379.
† "On the Arteries," p. 10.
‡ See Sir J. Paget on "Obstructions in the Pulmonary Arteries," "Medico-Chirurgical Transact.," vol. xxviii. p. 533.

The formation of a coagulum in the artery does not necessarily block up the entire passage, but may leave a central opening by which the circulation yet continues to be carried on. But after the formation of the clot, it in its turn undergoes various changes; it may become absorbed, or it softens or breaks up into granular matter, and is carried into the capillary circulation, or it is capable of organization, and we then find in it a network of fine blood-vessels, while perhaps most frequently of all it becomes tunnelled by the formation of a central passage. The last point serves to elucidate the observations of the passage of an artery occasionally seen in old coagula formed after the application of a ligature. Lobstein, as we are informed by Hasse, met with an arterial vessel of the calibre of the stylomastoid artery running lengthwise through the femoral artery obliterated two years previously by tying. Blandin and Barth have met with analogous instances, to which may be added those cases in which, after the complete obliteration of arteries by ligature, new vessels have been found shooting from their extremities. Arteries in which coagula have been formed at some previous time may thus be found lined by what looks like an adventitious false membrane; really resulting from the tunnelling of the once solid clot.

CHAPTER XXIV.

ANEURISM.

ANEURISM,* or a dilatation of an artery, is connected with two lesions, according to which, from the days of Scarpa and John Bell, downwards, two classes of aneurismatic disease have been adopted by most writers—true and false aneurism,—though we shall find grounds for assuming that spontaneous aneurism is in all instances traceable to one ultimate cause—a morbid state of the arterial coats, which may produce accidental varieties in the arterial tumour. The definition of true and false aneurism, ordinarily accepted, is that, in the former we have to deal with a dilatation, partial or entire, of a certain extent of an artery, without laceration of any of its coats; while in the latter, the dilatation is accompanied by the laceration of one or more of the coats.

The precisely opposite definition was, however, given by Scarpa (following Sennertus), who called a sac a true aneurism which was deficient in one or more of the coats; while if the wall was perfect, like that of the artery containing all three coats, he called it a false aneurism. The terms true and false applied to aneurisms have, therefore, no fixed meaning, and it is better not to use them.

Varieties of Aneurism.—We recognize the following forms of aneurism:—(1) *Diffuse*, produced by a general dilatation of the whole vessel uniformly in every direction, and over a considerable length, according to the amount of dilatation; this may produce a cylindrical or a fusiform aneurism. (2) *Circumscript* or *Saccular* aneurism, in which the whole circumference of the artery is not involved, but protrusion of part of it takes place, and a sac is formed on one side of the vessel, connected with it by an opening more or less patulous, which may be reduced to a mere pedicle. It is to the latter that Mr. Hodgson and some other writers attempted to confine the term aneurism. (3) *Dissecting* aneurism, in which, by rupture of the inner coat, the blood finds its way between the middle and outer, or among the layers of the middle coat. A somewhat similar condition is seen in the cerebral arteries, when blood may be effused within the outer coat, which in these arteries is loose, detached, and known as the perivascular or lymphatic sheath.

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* From ἀνευρύνω, I dilate; ἀνευρύσμα, a dilatation.

Those aberrant forms which are with doubtful propriety described by the same name are, (*a*) anastomotic aneurism, or tumor vasculosus arterialis, formed by dilatation and lengthening of a large number of minute arterial branches which communicate with one another. (*b*) Cirsoid aneurism, depending on the dilatation of an artery and some of its branches, which become tortuous and enlarged in a varicose manner.

CAUSES OF ANEURISM.

Arteritis.—It is now generally admitted that the chief factor in the production of aneurisms is disease of the vessels, viz., chronic endarteritis or the atheromatous process, which weakens the wall and favours its dilatation; but it is not quite clear why this extremely common condition so rarely leads to aneurism. It does not appear that this disease when it affects the inner coat alone can produce any conditions which weaken the resistance of the wall; but if it extend to the middle coat it produces a state of swelling and relaxation which must be very favourable to dilatation. In the later stages, when the coats are thickened, rigid, and even calcified, they would seem to possess increased power of resistance; and hence, in those cases in which we are able to trace the production of an aneurism, it appears to take place at an early stage of the atheromatous process rather than at an advanced stage. It has hence been referred to a specially "fibroid" change, which, however, we do not regard as distinct from the general process of chronic inflammation. It is not at all necessary that the walls should be thinner in order to be weaker. The condition in which there is thickening of the wall by the formation of soft, grey material (as described in the last chapter), especially when it affects the middle coat, appears to be the most favourable to the formation of aneurism. Aneurisms have also been sometimes regarded as due to constitutional syphilis by those who attribute chronic arteritis to this cause.

Pressure as a Cause of Aneurism.—It is impossible, on the other hand, to overlook the share taken by excessive vascular tension or strain in predisposing to aneurism. The disease is far commoner in men than in women; far commoner in men who go through violent muscular exertion than in those who live sedentary lives; and aneurism of the aorta or large vessels is hardly met with in young persons unless they have been exposed to some severe exertion. It is common among porters, sailors, and especially soldiers. Now all the causes which can be set down as producing excessive vascular tension may also be regarded as causes of chronic arteritis. It seems, therefore, impossible to determine whether the excessive pressure acts directly by causing dilatation, or indirectly by causing arterial disease which favours dilatation. At all events, the localization of the disease must be determined by the state of the vascular wall; while the excessive tension will act as a general predisposing cause.

Laceration of the Coats.—The laceration of the internal coat of the artery may occur at the early stages of the diseases, and be the first exciting cause of the aneurismal tumour. The aorta of a lady, whose case is detailed by Mr. Hodgson,* illustrated this mode of the formation of aneurism. The coats of the vessel were diseased, and presented at the arch a transverse rent, about an inch in length, which had penetrated to the middle coat. The blood had insinuated itself between the middle and external coats, the latter of which was elevated into a tumour, about two inches in circumference. A similar appearance was found in the body of George II. We see no difficulty in regard to this view of the occasional origin of aneurism. That the early stages should not often be presented to us in the dead subject, is easily accounted for by the rapid distension that will take place after the first laceration, and the equally rapid laminated deposit of the defensive fibrine. Rokitansky, however, denies this mode of origin altogether, stating that no such rent is ever detected. Another question is whether laceration of an artery ever occurs without some previous derangement in its coats, in what is termed the traumatic form of aneurism, without a penetrating wound. The extreme pliability and elasticity of the arterial system, compared with all the tissues that surround it, might alone suffice to answer the question; but the direct physiological experiments performed by Mr. Hodgson and Mr. Hunter, and Sir Everard Home,† as well as the pathological observations by the former,‡ positively determine the point in the negative. Mr. Hodgson states that he has repeatedly tried, in imitation of Richerand, to produce a laceration of the internal and middle coats of the popliteal artery, by violently extending the leg upon the thigh; but that he has never lacerated the coats of the artery unless the degree of violence was sufficient to rupture the ligaments of the knee, an event which certainly does not generally accompany those accidents to which patients attribute the origin of aneurism.

Embolism a Cause of Aneurism.—It has been lately made out that certain forms of aneurism, especially those of the cerebral arteries, are in some cases due to the previous existence of embolism, the immediate effects of which have passed away. The process seems to be as follows:—the artery obstructed by an embolus, at length becomes pervious again, but the wall at this point is left so permanently injured and weakened that it readily dilates into an aneurism. This explains the frequency of intracranial aneurism at special points, such as the middle cerebral arteries, or at bifurcations of the other arteries. Of course there are many such aneurisms without any history of embolism, but still there will often be a history of valvular disease accompanied by vegetations, fragments of which becoming detached might produce embolisms.* Spontaneous thrombosis may, of course, have similar consequences.

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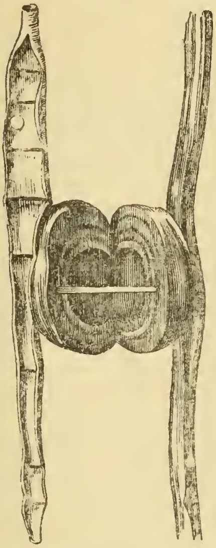
* "On the Arteries," pp. 39 and 63.

† "Transactions of a Society for the Improvement of Medical and Surgical Knowledge," vol. i. p. 144.

‡ "On the Arteries," p. 61.

Form and Contents of Aneurism.—The form of sacculated aneurisms is generally globular, but they may, partly owing to accidental conditions in the coats of the affected vessels, partly from the pressure exerted by surrounding tissues, assume an oval or more or less irregular outline. Upon the original aneurism, an evolution of secondary dilatations is sometimes met with, and these may even give rise to a further or tertiary multiplication of the disease, so as to induce a sort of mulberry appearance in the tumour; it is the variety to which Cruveilhier has applied the term "anévrisme sous l'aspect d'ampoules à bosselures."

FIG. 91.



Aneurism of the posterior tibial artery, with the nerve spread over the back part of the pouch; the sac is entirely obliterated by concentric layers of fibrine, growing paler towards the surface.

(St. George's Museum, F. 41.)

The contents of aneurismal sacs are fibrinous coagula, which form in successive layers, and accordingly present a concentric arrangement, like the annular rings in perennial plants. The resemblance may be traced still further in the gradual condensation of the outer or external layers, owing to absorption and compression. These also lose their colour, and become fawn-coloured or white; while towards the interior we continue to recognize the dark colour of the blood. The accumulation may proceed to such an extent as to obliterate the cavity, and thus establish a spontaneous cure. A considerable amount of organization is observed to take place in some deposits, manifested by the formation of fibres. The existence of a distinct membrane is assumed by most writers to envelope the coagulum. It is a point to which Bizot particularly drew attention, and upon which Hasse dwells forcibly. The latter states that he has never met with an aneurism in which this adventitious membrane was not present. Mr. Bowman has observed, that the membrane enveloping the coagula in an aneurism, though apparently of exactly the same nature as that lining the arteries, differs from it in not presenting any epithelium. This so-called membrane is in fact the condensed external layer of the coagulum, similar to that which forms on the surface of old clots in veins. It is not, properly speaking, organized. The coagula form in proportion as the sac is cut off from the rest of the circulation; the more shallow it is, and therefore the more exposed to the force of the current, the less the liability to the formation of fibrinous laminæ, and the less, we may also add, the coincident danger of perforation of the coats of the vessel. It is through the agency of this deposit that a spontaneous cure may take place, either by an obliteration of the sac, or by pressure upon the artery, and consequent obliteration of its channel. The coagula may subsequently undergo secondary metamorphoses, such as a conversion into cholesterine, or cretaceous matter. But they do not always form, and we may meet with a series of aneurisms on the same vessel, some of which present coagula, while others are empty.

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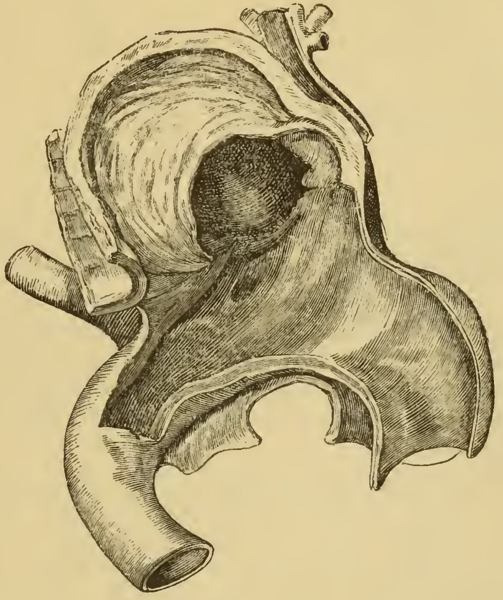
* J. W. Ogle: "Trans. Path. Soc.," 1857, vol. viii. p. 170; "St. George's Hosp. Reports," 1867, p. 285; "Medical Times and Gazette," 1866, vol. i. p. 196; see also Church: "Trans. Path. Soc.," vol. xx. p. 109; Semple: Ibid., p. 112.

Fig. 92.



Section of the arch of an aorta, with an aneurism arising from its upper part. The cavity of the sac is nearly filled by laminated coagulum, the internal membrane of the artery is thickened. The sac presses against the trachea, the arteria innominata, and the right carotid and subclavian arteries.

(St. Bartholomew's Museum, Series xiii. No. 11.)

Statistics of Aneurism.—In number and size there is a great diversity, as also with regard to the liability of different arteries to be affected, as well as in respect of sex. The following table contains an analysis of sixty-three cases, examined by Mr. Hodgson, which illustrates the last two points. It shows at once the great liability of the male sex, and the prevailing tendency of certain arteries to be affected * :—

	Males.	Females.	Total.
Ascending aorta, innominata and arch of the aorta	16	5	21
Femoral and popliteal	14	1	15
Inguinal	12	0	12
Descending aorta	7	1	8
Subclavian and axillary	5	0	5
Carotid	2	0	2
	56	7	63

Bizot's statistics agree closely with those of Mr. Hodgson as to the different liability of the two sexes; out of 189 analyzed by him, 171 occurred in men, and eighteen in women, which is even more in favour of the latter than Mr. Hodgson's table. The proclivity to aneurism is also determined by the age of the individual; it is unknown to childhood; the greatest tendency to the disease exists at the middle period of life, as shown by the following analysis of 108 cases :—

From 10 to 19 years	1 subject	From 50 to 59 years	14 subjects.
,, 20 ,, 29 ,,	15 ,,	,, 60 ,, 69 ,,	8 ,,
,, 30 ,, 39 ,,	35 ,,	,, 70 ,, 79 ,,	2 ,,
,, 40 ,, 49 ,,	31 ,,	,, 80 ,, 89 ,,	2 ,,

Dr. Crisp † found in 551 cases collected from journals, and 364 specimens from the London Museums, the following numbers :—

	Cases in Journals.	Museum Specimens.
Thoracic aorta	175	207
Abdominal aorta	59	46
Popliteal artery	137	50
Femoral artery	66	12
Carotid artery	25	9
Subclavian artery	23	12
Innominate artery	20	3
Axillary artery	18	8
Iliac arteries	11	10
Cerebral arteries	7	1
Other systemic arteries	8	4
Pulmonary artery	2	2
	551	364

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* The table is essentially the same as that given by Mr. Hodgson, but differently arranged. It excludes aneurisms arising from wounded arteries, and aneurisms by anastomosis.

† "Diseases of the Blood-vessels," 1847, p. 113.

Effects produced by Aneurisms.—As an aneurism enlarges it necessarily displaces the adjoining tissues, and causes an absorption of those that offer any resistance. The danger of an aneurismal tumour depends upon its site and upon its vicinity to vital organs whose functions are liable to be interfered with by pressure. It is thus that aneurism occurring in the thorax and in the regions of the neck threatens life, before the arterial disease has put on any dangerous appearance, by narrowing the trachea, by compressing the œsophagus or other vessels. The extent to which absorption prepares a passage to an advancing aneurism is in some cases extraordinary; an aortic aneurism by this process may pass through the thorax or eat into the vertebral column. In these cases, as Rokitansky describes, not only the bone is destroyed but the aneurismal sac itself becomes fused with the periosteum, and the other fibrous structures that usually invest the bones. In this way the exposed vertebral column may constitute a portion of the aneurismal wall. Sometimes we meet with a bone, such as a rib crossing the cavity of the aneurism, and attached only at the ends; the bone being at the same time denuded of periosteum. This paradoxical result is thus produced. The aneurism pressing on the rib causes absorption of the adjacent portion of periosteum and pushes the remainder off the bone, till the membrane forms part of the aneurismal wall; while the cavity, extending itself on both sides of the bone becomes continuous behind it.

Hodgson* has pointed out, that as the aneurism advances to the surface of the body, it induces sloughing of the integuments, and an eschar forming on the tumour itself, its discharge gives rise to fatal hæmorrhage; the same is the case when the aneurism opens into a cavity lined with mucous membrane. But a different result takes place when the sac projects into a serous cavity; in this case the membranes do not slough, but the parietes of the tumour become softened and thinned, and a laceration is effected. If a rupture of the internal and middle coats alone takes place, the external coat remaining entire, the blood may separate the latter to a greater or less extent without forming a sac; it then causes what has been termed by Laënnec, the dissecting aneurism.

Cure of Aneurism.—Nothing has been added by later writers to the observations of Mr. Hodgson on the subject of the spontaneous cure of aneurism, and we cannot do better than to extract his own terse summary of the subject: first, the whole tumour may be removed by sphacelation, in consequence of extreme inflammation excited by the distension of the surrounding parts; secondly, the tumour, as we have already had occasion to observe, may assume such a position as to obliterate, by its pressure, the superior or inferior portion of the artery communicating with the sac; and thirdly, the gradual deposition of fibrine in the sac and the artery leading to it, may render them impervious, and allow a subsequent process by which the tumour is removed. In the latter cases a gradual absorption of its contents takes place, the tumour becomes harder and smaller, and the establishment of a collateral circulation restores the balance of the circulation.

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* "The Diseases of the Arteries," &c., p. 85.

Distribution of Aneurisms.—Before quitting the subject of aneurism, we must allude to certain peculiarities in connection with its occurrence in different parts of the arterial system. We have seen that aneurisms are almost limited to arteries of the largest size; in smaller arteries, as in the radial and ulnar or tibials, they are rarely met with. They are altogether extremely rare in the upper extremity, they here almost invariably arise from carelessness in venesection, especially if, as on the Continent, a spring-lancet is employed. In such a case the result generally is a communication between the brachial artery and a cubital vein, especially the basilic, forming what is called varicose aneurism. Cases of spontaneous aneurism of the upper extremity are, however, occasionally met with. One recorded by Dr. J. W. Ogle is ascribed by him to the effects of embolism. The smallest arteries in which spontaneous aneurism is met with are the coronary of the heart and the cerebral. An instance of the former, which is extremely rare, is reported in the Records of the Pathological Society for 1848; it was discovered by Dr. Peacock in a man aged fifty-one, who had presented no symptoms of cardiac disease before death. The tumour occupied the left coronary artery, and was about the size of a pigeon's egg, containing lacerated coagula, which were intimately adherent to the lining membrane. There was some atheroma in the aorta. Dr. J. W. Ogle has collected a certain number of similar instances, in most of which the aneurism burst into the pericardium.[*]

Intracranial Aneurism.—Aneurism of the cerebral arteries, though not common, has of late been shown to be more frequent than was at one time supposed. It is generally seated at or near some part of the circle of Willis; it may attain the size of a walnut, and more, though it is commonly smaller; it is met with chiefly between the age of forty and fifty. Here, too, the male sex presents a much greater liability than the female; showing that the former manifestly possess a marked tendency to the disease, and that the increased tendency is not due to accidental circumstances. We find two good instances of aneurism of the cerebral arteries in the Reports of the Pathological Society, presented by Dr. Hare[†] and Dr. Roe[‡]; the one in the left posterior communicating artery, the other in the anterior cerebral; this one of unusual size, being as large as a hen's egg, had caused partial absorption of the sphenoid bone upon which it rested, and a flattening of the adjoining portions of the brain. Both the cases alluded to occurred in females; and

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[*] "St. George's Hospital Reports," 1867, p. 285. Dr. Crisp has made a similar collection of cases, "Trans. Path. Soc.," 1871, vol. xxii. p. 106.

[†] Report, 1849-50, p. 169.

[‡] Ibid. 1850-51, p. 46.

Dr. Roe's case was still further remarkable from its affecting the patient at the early age of twenty-one. Dr. Brinton,* from an analysis of about forty well-authenticated cases of cerebral aneurism, finds that three-eighths terminate in rupture, one-eighth from simple loss of functions by pressure, one-eighth by convulsive attacks, one-eighth by congestion or hæmorrhage of the brain, one-eighth by inflammatory conditions of the brain, and one-eighth by coincident disorders or accidents. In three instances Dr. Brinton found the aneurisms more than one in number; in one instance three were found; in one the opposite carotids were symmetrically affected.

These aneurisms sometimes occur in young persons, even at the age of thirteen, and when the arteries generally are healthy. Dr. Church † has collected twelve instances in persons under twenty, in the majority of which there were vegetations on the cardiac valves, and there is therefore a certain probability of their being due to embolism, especially as they generally occur at those situations on the arteries which are also often the seat of embolisms.

Visceral Aneurisms.—Aneurisms of the cœliac axis, of the mesenteric, hepatic, renal, and other visceral arteries are occasionally met with.

Aneurisms of the pulmonary artery and its branches are very uncommon, as is advanced atheromatous disease of these vessels. The only recorded cases have occurred in vomicæ produced by phthisis, and have often led to fatal hæmorrhage. The injury to, or inflammation of, the arterial wall caused by the breaking down of the lung tissue must be regarded as producing the aneurismal dilatation. Although observed as long ago as 1841 by Fearn,‡ they have been till lately almost overlooked, at least in this country. Dr. Douglas Powell § has found that such aneurisms or aneurismal dilatations exist in a large proportion of cases of fatal hæmorrhage in advanced phthisis.

Miliary Aneurisms.—Beside these larger aneurisms, we must again refer to the minute "miliary" aneurisms of Charcot and Bouchard,‖ before described as occurring in the brain. They are however not confined to the brain or meninges, though most numerous there, being occasionally found when searched for in other parts, as in the retina, on the surface of the heart, and in the submucous tissue of the œsophagus, intestines, and bladder.

Diffuse or Traumatic Aneurism.—The mechanical injury of an artery may cause the effusion of blood into the surrounding parts, which constitutes what has been termed diffuse false aneurism. It generally produces gangrene; but inflammatory reaction may be set up, and establish definite limits, and thus lead to the formation of an aneurismal sac. It is manifest that this, as well as the aneurismal varix, of which we have already spoken, has no pathological relation to the disease of which we have been treating.

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* Report, 1850-51, p. 48.

† "St. Barth. Hosp. Reports," vol. v. (1869), "Contributions to Cerebral Pathology;" Ibid. vol. iv. p. 142, Callender; Ibid. vol. iii. p. 433.

‡ "Lancet," Feb., 1841. Other cases in "Trans. Path. Soc.," vol. xvii. p. 79 vol. xviii. pp. 45 and 55, vol. xx. p. 105, vol. xxii. p. 37.

§ Ibid. vol. xxii. p. 41.

‖ Liouville: "Généralisation des Anévrismes Miliaires." Paris, 1871.

DISSECTING ANEURISM.

We sometimes meet with small ecchymoses under the lining membrane of the aorta in the dead body, which indicate the commencement of this form of aneurism. A minute and sometimes imperceptible fissure in the inner coat allows of the permeation of a small quantity of blood, and the first step having occurred, a succession of similar deposits may soon cause a greater accumulation, and necessarily a coincident separation of the coats.

True dissecting aneurism is not common, and is hardly ever seen except on the aorta. It arises from rupture of the inner and middle coats, and the blood after forcing its way between the middle and outer coats, forms a sac, limited externally by the latter, around the artery itself. This generally bursts externally, but has been known in rare cases to open again into the artery at some point lower down, so as to form a sort of second channel. The structures called dissecting aneurisms in the brain* are somewhat different; their peculiarities depend upon the special characters of the cerebral vessels. The outer coat or adventitia is here somewhat loosely attached, and a space is left between it and the other coats, which, being supposed to be connected with the lymphatic system, has been called a lymphatic space, while the adventitia has been called a perivascular sheath, names which have led to some confusion. Hæmorrhage, perhaps by "diapedesis," seems to take place very readily into this space in all hyperæmic conditions of the brain, and a small clot is formed around the vessel, but as there is no reason to believe that fluid blood remains in the space, or that the mass enlarges after it is once formed, it is plainly something very different from an aneurism. The blood effused soon degenerates or becomes absorbed, and a few yellow granules may alone remain as evidence of the hæmorrhage. The sheaths may doubtless become ruptured and produce more extensive hæmorrhage, but this does not seem often to happen. When hæmorrhage has already taken place the shock and destruction of tissue may produce very numerous perivascular hæmorrhages of this kind.

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* First described by Virchow and Kölliker.

CHAPTER XXV.

THROMBOSIS AND EMBOLISM.

THE fact of the coagulation of blood in the vessels during life is one of so much importance as to require special consideration. To this process the term *Thrombosis* (Gr. θρομβώσις, curdling or coagulation) has been applied; and a clot formed some time before death is called a *thrombus*, to distinguish it from those formed after death or when death is approaching. Such clots may be found in the heart, the veins, arteries, or capillaries; but they occur in veins far more frequently than elsewhere.

A thrombus is known from a post-mortem clot either from some changes having taken place in it since its formation, or else by a heterogeneousness of composition which shows that it was not all formed at one time; or possibly by some peculiarity showing that it was formed while the blood was in motion. In the absence of all these characters it is still possible that a clot may have been formed before death; but we shall not be able to prove it. The changes which take place in clots after their formation are such as adhesion to the walls of the vessel, rearrangement of their constituents, and either hardening and drying up or softening and liquefaction. The process called organization may be the final stage. The rearrangement of the constituents of the clot is shown by the centre becoming decolorized, and by the surface becoming covered with a whitish granular layer, composed chiefly of leucocytes. The latter change may occur without the others, and is of itself enough to show that the clot has existed some little time, *i.e.*, that it is a thrombus. Sometimes a clot is found to be distinctly heterogeneous or stratified, and composed of layers which are not wholly alike. This shows that it was not all formed at one time, and is, of course, positive evidence that great part at least was formed before death.

Varieties of Thrombi.—A thrombus which remains fixed at the spot where it was formed is called *primitive.* If coagulation spread gradually from this original point along the vessel we get a *prolonged* thrombus. In veins the process advances in the direction of the heart as far as the opening of the vessel into a larger vein; in arteries it more often spreads from a main trunk into the branches, but may also extend backwards towards the heart. Thus spreading it may either partially or wholly obstruct the vessel, which it will in the latter case completely fill. The end towards the heart will, in any case, be rounded or bluntly conical. *Secondary* thrombus is a term sometimes used for coagulation which is the result of inflammation; sometimes, and more properly when the coagulation spreads from one set of vessels to another, as from arteries to capillaries and veins, from capillaries to veins or arteries, &c.

Thrombi may also be divided into stratified and unstratified. The latter are formed by the sudden coagulation of all the blood in a vessel, as happens after ligature. The corpuscles of both kinds and other constituents will here be, at least in the first place, uniformly distributed. The latter are produced by slow coagulation in successive stages. On section they are found to be composed of layers, and to contain an unusual number of white corpuscles, strata of which alternate with strata of red corpuscles and fibrin. The layers are seldom perfectly parallel to each other or to the walls of the vessel.

Thrombosis in different Classes of Vessels.—Local coagulation of blood in the vessels requires special consideration according as it affects veins, arteries, or capillaries.

In thrombosis of veins coagulation of blood may be produced, (1) by a general cause; (2) by some special cause.

(1) The best ascertained general causes are retardation of the whole systemic circulation (as, for instance, from feeble action of the heart) and morbid alterations in the composition of the blood. It is easy to see how any obstruction of the general circulation must cause stagnation of the blood current in the veins, and so favour coagulation; this result is, however, not very often met with as a simple consequence of disease of the heart, but more often when the heart's action is enfeebled by some wasting disease; in which case other causes doubtless co-operate with the simple retardation. Such cases are with difficulty separated from those in which coagulation is ascribed to some change in the composition of the blood. Thickening of the blood from loss of its serum (as in cholera); increase in the number of white corpuscles (as leuchæmia); the more obscure changes caused by fevers as typhus, are all instances of such predisposing conditions. At the same time, we would point out that this explanation must be used with caution, for such a cause if actually operating ought to produce general coagulation, or at least simultaneous coagulation in several parts of the body; and even the latter is not very often observed. Another probable general cause not yet proved with certainty to be an actual one, is the introduction of some poison into the blood, or its generation there, as the hypothetical poison of pyæmia.

(2) Special or local causes are far more efficient in producing coagulation in veins. Of these pressure is the most common:

and a familiar instance is the binding of the arm after the operation of venesection. Pressure of tumours, inflammatory or indurative growths, abnormal pressure of the viscera themselves, as the pelvic viscera in females, or the abdominal organs in either sex, often lead to coagulation in the deep pelvic and abdominal veins. Dilatation or a varicose condition of the veins, by retarding the current, may have the same effect. Other morbid conditions of the walls are, in the case of veins, of very subordinate importance. Disease, especially inflammation in the organs from which the veins arise, frequently causes coagulation. The most familiar instances are thrombosis of the renal veins in cases of Bright's disease of the kidneys, and of the uterine or other veins in inflammation of the uterus or its appendages. Inflammation of the tissues surrounding a vein, or of the wall of the vein itself, is not less likely to produce the same result. Finally, we must not omit to point out a possible occurrence, once thought to be frequent, now known to be excessively rare; viz., the entrance into the vein of the contents of an abscess or other collection of morbid matter.

In all cases of thrombosis, the extension of coagulation to contiguous parts must not be disregarded. Thus thrombosis of a small tributary vein leads first to a projection of the clot into the cavity of the larger vein into which it opens, and then to the gradual formation round it of another clot which may finally completely block the larger vessel. Thrombi which block up the large venous trunks usually arise in this way. Moreover, obstruction of an artery, by whatever cause produced, may lead to coagulation in the capillaries supplied by it, and this coagulation be thus further propagated into the veins.

Thrombosis of Arteries.—Coagulation of blood in the arteries may be determined as in veins by pressure, by feebleness of circulation, or some other of the causes, special or general, acting from without, which were mentioned in the case of veins; but more commonly it is determined by the condition of the arterial wall itself. If we leave out of consideration the thrombosis which is a consequence of embolism, this is, in fact, by far the most frequent cause.

When by the process of chronic inflammation or atheroma, of which we have spoken elsewhere, the inner surface of the arterial wall becomes rough, it acts upon the blood contained in the vessel as a foreign body and causes its coagulation. In this way are formed in the larger arteries isolated masses of clot, which adhere to the walls. In smaller arteries it will be likely to cause complete obstruction; a result which may obviously occur subsequently in larger vessels. A specimen in the Museum of St. Mary's Hospital shows the descending aorta beset on its inner surface, with numerous flat, disc-shaped coagula, looking not unlike limpets adhering to the rock. These coagula were found to occur at spots where, by erosion of the lining membrane, calcareous plates lying in the arterial wall were exposed, and had caused precipitation of fibrin upon them. The cerebral arteries and the coronary arteries of the heart have been found blocked up by a similar process. It is easy to see how in all such cases a clot once formed will extend by causing coagulation of fresh layers of blood upon itself; and in this way spread through the whole diameter and length of one artery as well as into its branches. Obstruction seems, however, to be sometimes effected very suddenly by these means in the smaller arteries. Thrombosis of the coronary arteries of the heart has been known to cause sudden death; and thrombosis of the cerebral arteries sudden hemiplegia, or a "paralytic stroke."

In the greater number of cases, however, obstruction of arteries is caused by the impaction of a mass carried from a distance, or embolism, which we shall speak of directly. This may, of course, and often does, give rise to thrombosis, a clot extending in one or both directions from the embolus, after the manner of what we have called a secondary thrombus. The extent of this will depend upon the amount of collateral circulation, the situation of the branches, and also upon the rigidity or elasticity of the walls. If they are very elastic they will contract, forcing the blood out of them, and thus none will be left to form a clot; but if they are rigid, the vessels will not become emptied, and a long thrombus may be produced.

Thrombosis of Capillaries.—Coagulation in the capillaries of a certain area may be the consequence of obstruction either in the artery leading to the spot or the vein leading from it. It may also be the consequence of some condition operating in the blood, and is a constant occurrence in septicæmia or pyæmia. It has in these diseases been attributed by some to a chemical change in the blood, by others to the presence of minute organisms (bacteria), which block the capillaries.

Consequences of Thrombosis.—Thrombosis of veins produces chiefly œdema of the part from which the vein proceeds. Thus the feet and legs swell in the disease known as *phlegmasia alba dolens*, in which the femoral and iliac veins are coagulated. Thrombosis of an artery produces in the part supplied by the vessel a series of changes identical with those produced by embolism, and which may therefore be considered hereafter.

FURTHER METAMORPHOSES OF THROMBI.

Softening and organization are the most important modifications which thrombi ultimately undergo. *Softening* was at one time confounded with suppuration, but was shown by Mr. Gulliver, in 1839, to be really a distinct process; it has since been very fully studied by Virchow.* It occurs chiefly or solely in stratified thrombi, in which a limited portion of the centre becomes first pale, soft, and crumbling, and is then transformed to a thick, creamy fluid, much resembling pus to the naked eye. It contains, however, few leucocytes or formed elements, consisting chiefly of albuminous and fatty molecules. In rare cases something like actual pus, with abundance of corpuscles, is found. This process occurs frequently in thrombi of veins, and almost always in those of the heart; it often leads to the detachment of embolic masses.

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* Gulliver: "On the Softening of Coagulated Fibrin," "Trans. Med. Chir. Soc.," vol. xxii. Virchow: "Gesammelte Abhandlungen," 1856, p. 95, reprinted from "Zeitsch. für. Rat. Med.," 1846.

Organization occurs chiefly in homogeneous, unstratified thrombi in healthy vessels, more frequently in arteries than veins; it is the ordinary termination of thrombosis from wound or ligature. The thrombus becomes first of all very closely adherent to the vascular wall, it then partially loses its colour, and becomes in part converted into a yellowish mass, similar to the inner coat of the vessel. In this mass are subsequently formed blood-vessels and bands of connective tissue which completely replace the original mass, and themselves ultimately disappear or become reduced to very small dimensions. The processes of vascularization and conversion into fibrous tissue are somewhat complicated, and have been described in very different terms by different observers. O. Weber, with whom Rindfleisch agrees, attributed organization to the proliferation and development of the leucocytes in the clot itself. These were supposed to become united into a network similar to that formed by the corpuscles of connective tissue and their prolongations; while the red corpuscles and fibrin represented intercellular substance. The capillaries were believed to be mostly derived from the still permeable part of the vessel, a few connections only being formed with the vasa vasorum. It is as a matter of fact usually possible, when a thrombus is in process of being organized, to inject its capillaries from the main vessel; but the production of vessels has been of late explained in a very different way.

According to the latest observers, the organization of the thrombus is effected by changes in the epithelium of the vessel and the connective-tissue cells of the inner coat. Both these classes of elements proliferate within a few hours, and form spindle-shaped or fibro-plastic cells, which afterwards become transformed into vessels and connective tissue. As the thrombus becomes permeated with newly-formed capillaries the fibrin and corpuscles simply liquefy and disappear, so that the process is rather a substitution of newly-formed vascular tissue for the clot than a transformation of it. The young vessels become connected with those of the middle and outer coats of the vessel, so that a complete circulation is established, and subsequently enter into communication with the still open cavity of the vessel.

Other observers have attributed great importance to the activity of leucocytes, or migratory cells, which are supposed to make their way from the vasa vasorum, and from the blood still circulating in the vessel itself into the interior of the thrombus. It has, indeed, been shown that such a process actually takes place, though its importance is uncertain. It will thus be seen that though the organization of blood clot was traced long ago in its main features by Hunter, its minute characters are far from satisfactorily settled. Finally, either the walls become adherent all round, and the vessel is obliterated; or else more rarely an opening or canal remains, through which the circulation is carried on.

Canalization of Thrombi.—Clots which have become organized in the manner just described become perforated by openings, through which the circulation is re-established. These openings seem to be formed by absorption of areas between the newly-formed vessels and fibrous bands. A similar perforation, though not produced in the same way, is seen as a consequence of simple softening. In both cases the circulation may be restored, though probably the vessel never completely returns to its former state.

Calcification of thrombi is seen occasionally in veins. In such cases the clot is doubtless never organized, but the details of the process are not known. In this way are formed the phlebolithes, or vein-stones, which are not unfrequently seen in the deep veins and plexuses of the pelvic viscera, or less frequently elsewhere.

EMBOLISM.

By embolism * is understood the obstruction of vessels by masses brought from a distance and carried along by the blood current till they arrive at a channel too narrow to let them pass. It follows by definition that this process can only occur in arteries, capillaries, or in the divisions of the portal vein.

The objects which most frequently serve as emboli or blocks, are fragments of dislodged or crumbling thrombi from veins, similar fragments from another part of the same artery, or from the heart, and fragments of fibrin or vegetations detached from the valves of the heart. More rarely pieces of new growth, parasites, or foreign bodies. Emboli derived from the systemic veins of course pass through the heart into the pulmonary circulation, as do those derived from the right side of the heart; while blocks in the systemic circulation can only come from the heart, some part of the arterial system, or the pulmonary veins. Emboli of the portal system of course become arrested in some ramification of the portal vein within the liver.

The importance of the embolic process depends upon the nature and size of the obstructing mass, as well as upon the part of the body supplied by the obstructed vessel.

A mass of simple clot coming from a systemic vein may, if it be large enough, become entangled in the valves of the heart, or block up the trunk of the pulmonary artery, or one of its main branches, causing sudden death; if smaller it will interrupt the circulation in a portion of the lung, producing results to be presently discussed. Simple blocking of the systemic arteries will produce gangrene, hæmorrhage, dry necrosis, or other changes, according to circumstances.

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* Etymology from Greek ἔμβολος = a something thrust in, a plug or stopper; hence ἐμβολισμός = the process of thrusting in or plugging. But in English we often use embolism instead of *embolus* for the object itself, the plug.

On the other hand, a clot coming from a vein in a part of the body where there is inflammation, especially inflammation of a certain kind, may set up inflammatory and destructive processes in the part where it becomes arrested. This is the case in pyæmia, and hence the embolic process is a most important part of the machinery of pyæmia. Again, emboli composed of new growth may set up a similar secondary new growth in the parts to which they are distributed, and hence this process is one of the means by which the generalization of new growths is effected. The further consequences and symptoms of embolism will obviously depend upon the functions of that part of the body whose blood supply is cut off. The mechanism of the process has been chiefly explained by Virchow and Kirkes, but some points have lately been elucidated by Cohnheim.*

Secondary Changes in the parts affected with Embolism.—When a part of the body has its blood supply suddenly cut off, the following may be the consequences:—

1. No permanent effects follow if there is a sufficient collateral circulation by anastomosing arteries to furnish an adequate blood supply. In this case the only result is that the portion of artery intervening between the embolus and the next branch becomes obliterated. This may occur in blocking of the cerebral arteries before the circle of Willis, of the larger branches of the pulmonary artery, or of any arteries in the skin, muscles, digestive tract, glands, bones, joints, and other parts where the anastomotic arrangements are complete.

2. If, on the other hand, the artery is, in Cohnheim's words, "a terminal artery," that is to say, without any anastomosis, and communicating solely with capillaries, the blood supply is not restored, and the consequence is either (*a*) necrosis of the part supplied, or (*b*) engorgement followed by hæmorrhage, forming on a surface ecchymosis, in deeply situated organs infarctions.

The organs in which these processes are likely to occur are five; viz., the spleen, the kidneys, the retina, the brain, and the lungs, since in these "terminal" arteries are found.

(*a*) Simple necrosis occurs in organs secluded from the air when the secondary changes of engorgement and hæmorrhage are not superadded. It is seen most frequently in the brain; as, for instance, after blocking of the middle cerebral artery. In parts exposed to the air the necrosis becomes gangrene (if, as before, engorgement and hæmorrhage are absent). This is seen in blocking of an artery of the leg, if the collateral circulation be inadequate. It also occurs, though rarely, in the lung, where engorgement and hæmorrhage usually follow.

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* "Untersuchungen über die Embolischen Processe." Berlin, 1872.

(*b*) Engorgement and hæmorrhage are the usual consequences of blocking of an artery in the spleen, kidneys, retina, or lungs, and sometimes in the brain. Engorgement is caused by regurgitation of blood from the veins. When the blood current from the heart is stopped, and the vis à tergo of the blood current annihilated, the pressure from the capillaries into the veins becomes less than the backward pressure from the veins into the capillaries, till ultimately (provided there are no valves in the former) regurgitation takes place; which seems to occur very slowly and gradually. The capillaries, and even arterial branches, become in this way engorged with venous blood, and the whole region is, as was before pointed out ("General Pathological Anatomy," p. 58), in the condition of venous hyperæmia. How this condition passes into hæmorrhage has only recently been fully explained by Cohnheim.

It is difficult or impossible to conceive that hæmorrhage could be caused merely by excessive tension, since the pressure caused by regurgitation could hardly be greater than the normal arterial pressure. Cohnheim explains it to be a consequence of the alterations in the structure, or, in fact, necrosis of the walls of capillaries caused by the privation of arterial blood; for venous blood cannot, as is well known, serve for the nutrition of the tissues. This was established by experiments on the tongue of the living frog, in which an arterial branch was shut off by a ligature, and after a while the blood readmitted. General hæmorrhage then took place from the capillaries. Thus in the region of an embolus, the regurgitant venous blood comes into a system of capillaries, the walls of which are already injured or necrotic from the absence of arterial blood, and which accordingly permit extravasation of red corpuscles. (This must not be confounded with emigration of leucocytes in inflammation.) From this explanation it follows that a certain time is necessary for the production of hæmorrhage; and, in fact, we find that in the case of a quite recent embolus, these changes are not observed.

The region thus affected with engorgement and hæmorrhage presents a peculiar appearance, that of the so-called hæmorrhagic infarctus or "block," which may be summarily described as a wedge-shaped or pyramidal mass,—the base situated towards the outer surface of the organ, the apex internally—of a blackish red colour, hard consistence, and projecting a little above the level of the surrounding parts. These peculiarities depend on the capillaries being gorged with blood (which ultimately coagulates), and the tissue being infiltrated with extravasated blood. They can only be seen in organs which contain terminal arteries, and veins without valves; commonly only in the spleen, kidneys, and lungs, and but rarely in the brain.

Variations are produced by accidental circumstances, such as position, influencing the venous circulation, the force of the heart, the possible coagulation of blood in the veins, and the like. It should not be forgotten that total obstruction of an artery by thrombosis will have the same consequences as when it is produced by an embolus. Partial obstruction appears to produce gradual necrosis rather than hæmorrhagic infarction. In the retina the shape of the organ and the arrangement of the vessels lead to a superficial ecchymosis being produced instead of a wedge-shaped block.

Subsequent Transformation of the Hæmorrhagic Infarctus—Engorgement and hæmorrhage do not avert, though they may delay, the necrosis which is the necessary consequence of interrupted nutrition. Hence a hæmorrhagic block passes through a series of necrobiotic changes ending with absorption. The central parts become pale, crumbling, and ultimately soft; and this change extends towards the periphery; on the other hand, the parts immediately surrounding the block become inflamed, and fibrous tissue is produced, which may form something resembling a capsule. The changes much resemble those of actual thrombi. The central parts may soften into puriform matter, but true suppuration does not occur; ultimately nothing remains but a cicatricial mass of newly-formed tissue, which by its contraction and shrivelling usually forms a depression on the surface of the organ.

CHAPTER XXVI.

DISEASES OF THE VEINS.

INFLAMMATION OF VEINS.

This condition was once thought to possess a much greater importance than we now assign to it, since it was regarded as the chief cause of coagulation in the veins; and, in fact, the term Phlebitis was used to mean precisely what we have elsewhere (following Virchow) called Thrombosis. The two phenomena, inflammation and coagulation, are very frequently found occurring together, but it is now known that in nearly all such cases, the coagulation determines the inflammation. It is, however, often impossible, at the stage at which the phenomena come under our observation, to decide the question of priority.

Primary inflammation, or phlebitis, may be acute or chronic.

Acute phlebitis is produced by direct injury, by inflammation in surrounding parts, by perforation of the wall by pus or sanies, or especially by coagulation within the vein. The inflammation produced by these causes is chiefly developed in the middle and outer coats, forming the mesophlebitis and periphlebitis of Virchow. Endophlebitis, or inflammation of the inner coat, is hardly seen in an acute form, except as a consequence of the coagulation of blood in the vein. Since as, in arteries, the middle and outer coats are the only vascular parts, they will naturally be most readily affected by inflammation, and show its phenomena most distinctly.

There is vivid injection of the outer coat, sometimes with extravasation; and less pronounced redness of the middle tunic, which may be visible through the inner. The walls are much swollen, thickened, and infiltrated with serous or purulent exudation. The cavity becomes at the same time dilated from paralysis of the muscular fibres, so that the vein altogether appears greatly enlarged. The inner coat becomes dull, opaque, easily torn, and sometimes flakes off spontaneously in continuous pieces. Coagulation of the blood contained in the vein is always found when the inflammation has reached this stage, and is doubtless sometimes produced by phlebitis, though, as has been said, the converse is usually the case.

The condition of the thrombus generally corresponds in some degree to the condition of the wall; so that when the latter is excessively thickened and infiltrated with purulent matter, the thrombus will be breaking down or in the condition of puriform softening.

Endophlebitis.—While in many cases the changes just described may proceed without participation of the inner coat, in other cases important changes are seen in the latter. Abundant proliferation of the endothelial cells takes place, producing either continuous thickening or budding masses of new growth which penetrate the softening thrombus. Durante * found when a single ligature was put round a vein, the inflammatory changes were most conspicuous in the intima; but in a portion of vein enclosed between the ligatures the latter membrane remained passive, while all the processes of destruction and organization went on in the other coats.

These changes have, however, been traced by several other observers in inflammations produced experimentally; and there is no doubt that they play an important part in the apparent organization of thrombus and the obliteration of veins after ligature.†

Bubnoff ‡ has shown that migratory cells or leucocytes may penetrate the venous wall from outside, and has attributed to the influence of these elements a large share in the changes just described.

Chronic Phlebitis.—Chronic inflammation of the outer coats occurs in all probability as the sequel of acute inflammation and thrombosis. The vein is found with its walls, especially the outer coat, thickened, and very closely adherent to the surrounding connective tissue, which is also indurated. The middle coat also shows fibrous thickening; and there may be thickening of the inner coat, so that the whole wall is greatly hypertrophied. The vessel is often dilated.

Chronic endophlebitis resembles the corresponding process in arteries, but is comparatively uncommon. It is seen chiefly in the large veins, when the inner coat may be found yellowish opaque, thickened, and rigid.

Puriform Thrombosis and Phlebitis.—When the process of thrombosis occurs in parts which are the seat of suppurative inflammation, it may, under conditions very imperfectly understood, assume the character of what has been called suppurative phlebitis, or suppurative thrombosis. The clot softens and breaks down in such a way as to resemble suppuration, which indeed is said in some cases actually to occur; perhaps from the passage of pus cells through the walls into the interior of the vein. Secondary phenomena of the most destructive character result from this process. In this case a soft, straw-coloured spot forms in the centre of the coagulum, the lamination of the latter disappears, until the whole is converted into a grumous mass. When portions of this mass are propelled into the circulation, the symptoms of poisoning result, and we find particles of the morbid product becoming arrested in the pulmonary circulation, and there occasioning coagulation of the blood, or the formation of abscesses. Coagula resulting from this cause are most commonly met with in the right side of the heart, and in the distribution of the pulmonary arteries. They are laminated, and are softened in the interior, where there may be a nucleus of puriform matter. The introduction of these matters into the circulation is generally accompanied with symptoms of a general prostration of the powers, of the most intense character, which are due rather to the infection of the blood, or pyæmia, than to local results in the shape of lobular abscesses, or secondary purulent deposits in distant viscera. The local results are doubtless due to the infective properties of the transported masses, since simple emboli do not produce at all the same changes; and these properties probably depend upon their being imbued with some poison from the original seat of inflammation. It is thus one of the forms of pyæmia; but an affection of the veins is by no means a necessary part of that disease.

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* Stricker's "Jahrbücher," 1871-72, p. 143.

† Cornil and Ranvier: "Hist. Path.," p. 570; Thiersch in Pitha and Billroth's "Handbuch der Chirurgie," vol. i. division ii.; Waldeyer, "Virch. Archiv," xl. p. 369.

‡ "Virch. Archiv," xliv. p. 462.

Authors of our own country were the first to trace the connection between secondary deposits and local injuries. Morgagni and Desault had particularly alluded to abscesses in the liver following cerebral lesions, but it was reserved for Arnott * and Davis † to show the actual physical connection between these occurrences, and to establish the real nature of the so-called phlebitis. The latter was the first to demonstrate that it is the pathological phenomenon constituting phlegmasia alba dolens, a malady accompanying the puerperal state, which had previously been attributed to a reflux of the lochia, to milk depôts, or to obstruction of the lymphatics. He showed, by a post-mortem examination of four fatal cases, that it resulted from "inflammation" of one or more of the principal veins within, and in the immediate neighbourhood of, the pelvis, producing an increased thickness in their coats, coagulation of their contents, and occasionally a destructive suppuration of their whole texture. Many cases of puerperal fever are mainly due to infective thrombosis of the veins of the uterus, and secondary inflammations induced thereby. This appears to have been more particularly the case with the frightful endemic of puerperal fever, which used, until very recently, to rage in the great hospital of Vienna. Dr. Semelweiss made the discovery that it mainly was due to the introduction of poisonous matter into the vagina and uterus of parturient women, by the medical men and students, who had recently been handling post-mortem specimens. The proper

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* "Medico-Chir. Trans.," vol. xv. p. 1.
† Ibid. vol. xii. p. 419.

precautions that have since been adopted have almost banished the disease.* Thus, while in 1846 there were 459 deaths among 3,354 females, in 1848, after the employment of chlorinated solutions by the medical attendants for their own purification, had been introduced, the deaths in 3,356 patients had sunk to the comparatively small number of 45. In many cases of phlebitis and pyæmic infection resulting from venesection, the effect has been traced to a similar cause, viz., the introduction of poisonous matter into the vessel, either by the lancet, or by the use of foul sponges. It was from its occurrence after phlebotomy that the attention of John Hunter was first drawn to the subject.

For a long time the liver was supposed to be the only organ in which metastatic abscesses, as secondary suppurations used to be called, were found, and then only in connection with injuries of the brain. It was first shown by Arnott that no organ of the body is exempt from this lesion, though it occurs more frequently in the liver and the lungs, and next in order in the kidneys, the spleen, the heart, and superficial tissues, while it is met with but rarely in the brain and the cavities of the eye. The abscesses vary in number, but when found in one organ, we may expect to find them in others also, and it is rare to meet with a solitary secondary deposit. Thus, in a case that occurred at St. Mary's Hospital, in a boy, in whom infective thrombosis and phlebitis were brought on by an accidental contusion, and death occurred within six days of the injury, and three of the occurrence of any alarming symptoms, secondary abscesses were found in the lungs, the heart, and the kidneys. Velpeau† relates the case of an individual, in whom from fifteen to twenty abscesses were counted in the brain, from eight to ten in the lung, and purulent deposits were also found in the kidneys, the spleen, and the liver.

In addition to the cases of thrombosis and inflammation of individual veins, already treated of, there are others to which it is right that we should especially advert. An inflammation of the umbilical vein in infants is mentioned by Kiwisch,‡ as occurring almost epidemically; and Dr. Lee has also met with it coincidently with the epidemic occurrence of metrophlebitis. The affection generally commences between the second and fourth days, and is followed by peritonitis and icterus. In the adult we meet with inflammation of the vena portæ, which may occur idiopathically, or by extension of inflammation from the mesenteric veins. It has been seen resulting from a fish-bone penetrating through the coats of the stomach, into the superior mesenteric; and the cases in which no such lesion could be discovered have been set down to metastatic irritation, to the irritation produced by spirituous beverages, suppressed hæmorrhoids, gout, or erysipelas. The occurrence is marked by the appearance of what exactly resembles the formation of numerous abscesses in the substance of the liver, but which, on close examination, prove to be accumulations of pus or puriform matter in the branches of the vena portæ. The cases of this disease that are recorded are so few that we are induced to quote the following instance, which fell under our own observation; it is the more remarkable as it presents the only instance of apparently idiopathic ulceration of the trunk of the vena portæ that has been published. It occurred in J—— W——,* a labouring man, a patient of Dr. Alderson, at St. Mary's Hospital, who, seventeen days previous to admission, was seized with a shivering fit; since then he had suffered from rigor, followed by heats and perspirations, at irregular intervals. On the 1st of October, 1852, a fortnight after admission, the skin is first reported to have been somewhat jaundiced, the pulse eighty-eight, tongue coated, loss of appetite, a burning sensation at the top of the sternum, with great depression of spirits. The yellow tinge of the skin continued, and the fits were fewer in number. On the 11th, the dulness of the hepatic region was found increased, and the stools are noted to have been dark. On the 18th, the skin was less yellow, the percussion of stomach and colon was tympanitic—no pain—no increase in the hepatic dulness. The shivering fits now returned more frequently, pleuritic symptoms supervened, the patient became more and more weak, more jaundiced, and drowsy, and sank on the 24th of October, 1852. In the thorax, the deposit of fresh lymph on the lower edge of the right lung was all the evidence of

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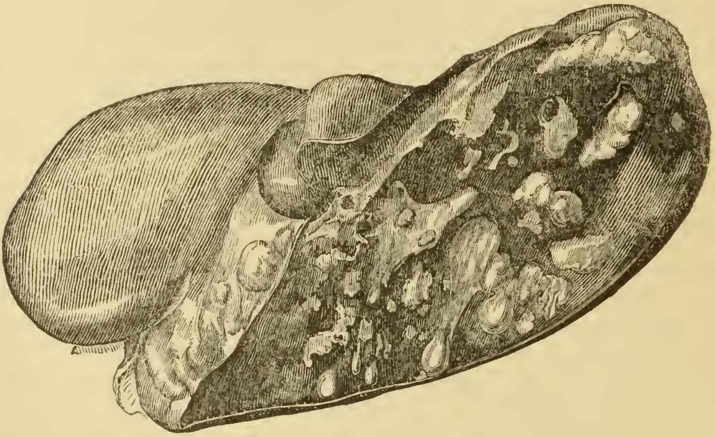
* See Dr. Routh on the "Endemic Puerperal Fever at Vienna," "Med.-Chir. Trans." vol. xxxii. p. 27.

† "Revue Médic.," 1826, vol. x. p. 442.

‡ "Die Krankheiten der Wöchnerinnen," Prag. 1840, vol. i. p. 112.

Fig. 93.



Section of liver exhibiting the appearances presented in inflammation of the vena portæ.

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* The case is abridged from the records of St. Mary's Hospital.

recent disease to be found. The lungs, the heart, are noted as being healthy, so also the kidneys and the brain. The liver was found much enlarged and dark coloured, feeling soft at many points; on removing it from the body, about two or three ounces of purulent matter escaped from the portal vein; the organ, at the same time, shrinking under the hand. On incising the liver, numerous bright yellow circumscribed spots appeared, closely resembling abscesses; they varied in size from a pin's head to a walnut. They proved to be all connected, occupying the ramifications of the portal vein, gorged with pus, of a perfectly laudable appearance. On examining the portal vein, it presented close to the point at which the splenic and superior mesenteric meet, a puckered, ulcerated appearance of its inner surface, extending for about an inch towards the liver; the ulceration was found to have penetrated through the inner coat, the edge of which was turned up, and well defined towards the healthy part of the vein. The ducts and the hepatic vein were found healthy, as also the orifices of the mesenteric and splenic veins. The surface of some of the portal branches was smooth, and these only seemed implicated as receptacles for the pus, while, in others, the lining membrane was destroyed, and a layer of puriform matter substituted. The hepatic cells proved everywhere natural, the lobules were loaded with yellow pigment in the middle, and there was marked hepatic venous congestion, which, it may be observed, closely resembled congestion of the inter-lobular plexuses. The intestines were only partially examined, but so far appeared healthy. This case contradicts the assertion of Rokitansky, that inflammation of the vena portæ invariably induces purulent and ichorous abscesses in the liver, and abscesses in the lung, with a very highly developed pyæmia. No abscess was discovered either in the hepatic or pulmonary tissue, nor was there any post-mortem evidence of pyæmia, for the suppuration was limited, in the most remarkable manner, to the trunk and branches of the vena portæ.

In the cranium we meet with similar changes in the sinuses of the dura mater, as a consequence of direct injury, and not unfrequently as a result of otorrhœa and caries of the petrous portion or mastoid cells of the temporal bone. Cerebral thrombosis is necessarily commonly associated with meningeal inflammation. It appears that the sinuses of the dura mater are liable to a chronic form of inflammation in children, leading to their obliteration and conversion into fibrous cords. Such a condition has been found by Tonnellé and by Gintrac, in cases marked by symptoms of cerebral congestion and apoplexy. The latter author * gives the following case in illustration of this condition; a child, aged four years, was liable from its birth to a temporary suspension of voluntary movement. There were no premonitory symptoms, and the attack occurred equally in the erect or recumbent position; the

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* "Recueil d'Observations," Bordeaux, 1830. Quoted by Andral, "Clinique Médicale," tom. v. p. 266.

intellectual faculties were maintained, but the power of articulation was suspended. The child died of pneumonia, supervening upon variola. The post-mortem examination exhibited the superior longitudinal sinus converted into a hard cord, the veins in connection with which were filled with coagulated blood. The walls of the sinus were thickened, dense, and of a yellowish colour; it contained a solid clot; no further lesion was discovered in the cranium.

Among the local cases of phlebitis not followed by general infection of the blood, those of the hæmorrhoidal veins are the most frequent, though it is a complication much to be feared in all operative proceedings directed to their cure, as well as to that of varicose veins of the legs or spermatic cord.

RUPTURE OF VEINS.

Rupture of the large veins is an event very rarely met with; Haller* quotes a case of rupture of the vena cava inferior, attributed to eating ice; but most of the instances recorded † were brought on by mechanical injury. The rupture of smaller veins often occurs as a result of sudden and forced distension. Thus small ecchymoses are frequently brought on in the conjunctiva by violent coughing. The veins of the lower extremities have been found ruptured by spasms of the muscles of the calves. The hæmorrhage that constitutes epistaxis, the menstrual, and hæmorrhoidal discharges, is rather analogous to the process of exosmosis, than a result of actual rupture, and therefore is rather more immediately connected with the capillary, than the venous circulation. A spontaneous rupture of larger veins occasionally results as a secondary consequence of varices. The sanguineous tumours in the labia of pregnant and parturient women are attributable to this cause, as also the laceration of varicose veins of the extremities, with or without coincident ulceration.

DILATATION OF VEINS.

Dilatation or varicosity of the veins is a subject which has attracted the attention of pathologists from the days of Hippocrates, who already distinguished between two kinds, which he termed hæmorrhois and cirsus. The affection is also treated of by writers under the generic term of phlebectasis. It consists mainly in an enlargement of the calibre of the vessels, and may or may not be accompanied by an alteration in their coats. Briquet ‡ avails himself of these differences for establishing his classification. He assumes three varieties—simple dilatation; uniform dilatation, accompanied by thickening of the coats; and irregular dilatation, with thickening or attenuation. The distension is generally owing to some impediment being offered to the return of the blood to the heart; and we therefore most commonly meet with it in parts in which the surrounding tissues are lax, and consequently do not offer a sufficient resistance to the pressure of the blood. Hence varicose veins are most commonly met with in the vicinity of the rectum and pudenda, and in the lower extremities. The veins swell, and assume a nodulated appearance and tortuous course, while the increased local pressure gives rise to an hypertrophy of the coats. Gendrin [*] and Briquet attribute the latter to chronic inflammation. A necessary consequence of the dilatation is an insufficiency of the valves, which no longer close the passage to the regurgitating current. They suffer a solution of continuity, and may become partially or wholly obliterated. The occurrence of phlebectasis has been ascribed to a peculiar constitution, which Hesse terms a morbid predominance of the venous system, a venous habit of the body, which may be characterized as one of general laxity of fibre and want of tone, associated with a tendency to local congestions. Age exercises a marked influence upon the occurrence and prevalence of the affection. It rarely manifests itself in any form until puberty, and is most common during the prime of life, as the tendency to it gradually ceases with advancing years. An hereditary predisposition may very frequently be traced. There is also a marked difference in the two sexes in regard to their proclivity to certain forms of the disease. Thus the hæmorrhoidal form is more frequently met with in the male sex, and its symptoms in many instances have suggested the impression that it is an analogue to the menstrual secretion in the female, from the periodicity of its occurrence and the relief the flux affords to the system. In some rare cases, recorded as curiosities by various authors,[†] a dilatation of the large veins in the cavities of the trunk has been observed. We must content ourselves with alluding to the fact, and pass to the consideration of the ordinary forms of the disease.

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* "Elementa Physiol.," vol. i. p. 130.

† James Kennedy has collected all the known cases of rupture of the vena cava inferior in "London Medical Repository," vol. xx. 1823.

‡ "Histoire des Inflammations," vol. ii. p. 9, et seq.

Varicocele or Cirsocele.—This disease affects the male sex commonly at the commencement of puberty. It consists in a dilatation of the veins of the spermatic cord, and prevails more on the left than the right side—a circumstance attributed to the more circuitous route taken by the left than the right spermatic vein. How rarely it affects the right side is shown by the fact, that in one hundred and twenty cases operated upon by Breschet, all but one occurred on the left. It is important, on account of the atrophy of the testicle which it is likely to induce, from its causing hæmatocele, by hæmorrhage into the tunica vaginalis, and from the influence which, in common with all sexual diseases, it exerts on the mind of the patient. The form of varicosity in the female sex, corresponding to varicocele in man, is enlargement of the vaginal and pudendal veins, which, especially during the advanced periods of pregnancy, are the cause of much suffering, and may, during labour, give rise to very severe hæmorrhage.

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[*] "Archives Générales de Médicine," vol. vii. pp. 200 and 396.

[†] See "Puchelt das Venensystem," vol. ii. p. 378, et seq.

Hæmorrhoids.—Hæmorrhoids, or piles, were formerly thought to consist in an enlargement and varicose condition of the veins surrounding the anus, but are now known to be more complex in their structure. They may occur in terminal branches of the inferior mesenteric, a tributary of the portal vein, or of the internal iliac. They protrude in the form of bluish nodes, or form flat sessile tumours. From their position, and the frequent pressure and congestion they are subject to when once formed, they are liable to inflammatory attacks; in consequence of which the surrounding cellular tissue condenses and hardens. Small cysts are formed in the latter, into which blood is effused, and they then exhibit a complex structure, which has been the source of much disputation. Abernethy and Kirby even went so far as to deny that they were owing to varicosity of the veins, and asserted them to be mere sacculated prolongations of the condensed submucous tissue. They are, in fact, composed of convolutions of tortuous and varicose small veins, forming a sort of cavernous tissue by intercommunication; but may be combined with simple dilatation of larger veins. The sequelæ to which they give rise are hæmorrhage, ulceration, and prolapsus of the rectum. The periodical character that is often observed in the sanguineous flow, is one that peculiarly deserves the attention of the physician. In man the affliction is common in persons of the middle of life, who have followed a sedentary pursuit; in women they are more apt to occur during pregnancy, and as a substitute for the menstrual discharge at the period of the climacteric.

Varicose Veins of the Leg.—Of all forms of varix, none is, probably, of so frequent occurrence as that which affects the superficial veins of the lower extremities, and more particularly the ramifications of the saphena. It is not peculiar to either sex, but is decidedly more common in females than males. This remark does not appear to apply to the Continent, however, for we learn from the statistics of Briquet, as well as from the statements of Hasse, that, with them, the male sex is the most liable. Another statement of Briquet, that it is more frequent in the right than in the left leg, is not confirmed by British experience. Hasse observes that, in men, the dilatation generally arises from the trunk, or the principal branches of the saphena, while he states that, in women, it commences in the minute twigs. It is especially at the ankle, and at the inner side of the popliteal space, that the veins are seen and felt, in the shape of an accumulation of tortuous vessels, of a more or less resistent feel.

A varicose state of the veins of the pia mater is a condition upon which Rokitansky lays some stress, as found after repeated attacks of delirium tremens. Oculists treat of dilatation of the veins of the eye in various forms; and instances of varix in other parts of the upper half of the body are recorded by authors. Thus, Cruveilhier * delineates two cases of varix affecting the arm.

Varicose veins may prove dangerous, by giving rise to hæmorrhage, in consequence of ulceration or rupture. They are not like the arteries, subject to atheromatous disease, though occasionally they become obliterated by the formation of a coagulum, or spontaneous inflammation and cohesion of the parietes.

OBSTRUCTION AND OBLITERATION OF VEINS.

An obliteration of portions of the venous system, from spontaneous coagulation of the blood during life, is a not unfrequent occurrence, either from the pressure exerted by morbid growths, as aneurisms or cancerous tumours, or without such mechanical causes, from some cachectic condition, as, for instance, in a case of empyema that fell under our notice, in which, from the inferior cava downwards, the veins were plugged up with a fawn-coloured coagulum. They are not, however, limited to the veins of the inferior half of the body, though most frequently met with in the vena cava inferior, and the portal vein. Dr. Bright † records a case in which the longitudinal sinus was filled in a child of twenty months, another of a female aged seventeen, in whom the left jugular and subclavian veins were plugged with a firm coagulum, terminating abruptly just as they entered the cava, and a third, in a female aged twenty, in whom a white, fibrinous coagulum was found in the subclavian vein, extending two or three inches up the jugulars. In both the last cases the hardened veins were traced during life; the subjects were all in an extreme state of exhaustion. In these cases the coagulum can be easily removed from the channel of the vessel, and the coats of the latter present no evidence of inflammation in the shape of thickening or interstitial deposit, or roughening of the lining membrane. Another form of obliteration is that resulting from a chronic inflammation, and consequent adhesive process, set up by the advance of degenerative disease, such as tubercle or cancer. Thus Dr. Lee records two cases of abdominal phlebitis resulting from malignant disease of the uterus; and another of inflammation of the iliac veins in a man, from carcinoma, is related by Mr. Lawrence.

NEW FORMATIONS.

Tubercle never directly affects the vessels; it is not found deposited in the coats, nor is it found in their channels.

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* "Anatomie Pathologique," livr. xxiii. and livr. xxx.

† "Medical Reports," part ii. pp. 60, 64, 65.

Carcinomatous and allied growths, on the other hand, are very frequently discovered within the veins as an immediate extension of the disease, external to them. This may happen by actual perforation of the venous wall, but in other cases a sort of infiltration takes place, the wall remaining entire. The growth may also arise in a thrombus formed within the vein, without any continuity with the mass outside. Thus in cancer of the stomach and liver, it has been found in the vena portæ; in renal cancer, in the corresponding vein and the vena cava inferior; in uterine cancer in the vena cava and its branches. Few of the observers of the cases on record have verified the fact of the cancerous nature of the contents of the vessels by microscopic examination, and in many instances fibrinous coagula have been mistaken for cancer, when coincident with the latter.

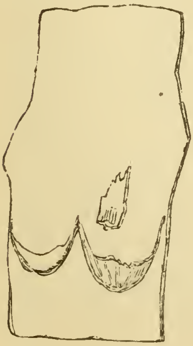
We have, however, on several occasions established the presence of actual carcinoma in the renal veins when that disease affected the kidneys, and of a round-celled sarcoma in the portal vein when a similar growth connected with the head of the pancreas surrounded the vein. The new growth may entirely fill the vessel, and sometimes fragments detached from it may form emboli, which being arrested in the lungs may set up secondary growths there.

New formation of connective tissue takes place as a thickening of the wall, but never as an isolated tumour.

Almost the only form of simple tumour known to affect veins is myoma, which has been occasionally observed.

Vascular tumours, or angiomata, often proceed from veins.

Fig. 94.



Calcareous deposit in the coats of a vein. (St. George's Museum, F. a. 12.)

Calcareous Deposit.—Ossification very rarely affects the veins, but cretaceous deposits are occasionally discovered under the lining coat, as in the case of which we have given a delineation. Cruveilhier relates the case of an old man who died of gangrena senilis, in whom the veins accompanying the popliteal artery were studded with phosphatic deposit.

Phlebolithes.—The concretions termed veinstones or phlebolithes, are met with free in the cavity of the vessels. They are formed of concentric laminæ, of which the internal are hard and brittle, while those forming the outer layers present a softer consistency. They closely resemble the concentric corpuscles so frequently met with in the choroid plexus, where, however, the formation is external to the vein. They are found most frequently in the pelvic veins, and in varices, and appear to result from a stasis in the blood, first giving rise to a coagulum of fibrine, within which a process of cretification takes place; chemically they are found to consist of phosphate and carbonate of lime, bound together by animal matter. The theory of their formation agrees with the mode of explanation which suggests itself for other concentric corpuscles, and is confirmed by what we occasionally see in diseases. Thus in a case of a large cyst in the kidney, containing within an inner sac of false membrane a large black coagulum of blood, we found concentric corpuscles in the false membrane, of exactly the same character as those observed in the brain. Here, too, it seemed reasonable to assume the primary deposition of fibrine, and the precipitation within its laminæ of the phosphate of lime. It is not impossible that phlebolithes may in some instances be the residuary traces of former phlebitis. Dr. Lee observes,* that in the spermatic and hypogastric veins of females advanced in life, calcareous concretions and disorganizations of various kinds have frequently been observed, which must have been the consequence of attacks of acute inflammation at remote periods.

ENTOZOA.

Before quitting the pathology of the veins, we have to allude to the presence of entozoa, and of gaseous contents within them. Of the former, instances are recorded by various of the older authors, from Pliny the Elder downwards. This writer states, in his "Historia Naturalis," that animals form in the blood of man, and destroy his body. An observation of parasitic animals in the blood is recorded by Dr. Bushman; † but it is liable to objections which tend to invalidate the conclusions arrived at. The observation of the presence of distoma hepaticum in the trunk of the vena portæ in a man aged forty-nine, by Duval,‡ is more valuable and trustworthy. Andral § recounts an instance of hydatids found in the venous system. They occurred in the pulmonary veins of a man aged fifty-five, and twenty-three occupied the small ramifications shortly before their transition into the capillary network. They varied in size from a pea to a nut, and were symmetrically distributed through both lungs. They had all the characters of acephalo-cysts. A similar observation was made by Wunderlich. Of late years the distoma hæmatobium has been observed in the blood in cases of the endemic hæmaturia met with at the Cape and in other parts of Africa; and a species of filaria has also been found in India, producing the disease called chyluria.

AIR IN THE VEINS.

The entrance of air into the veins is one of the most formidable occurrences complicating operations about the neck that the surgeon has to deal with. Death ensues rapidly, and atmospheric air is found in the right side of the heart. Air has been traced in some of these cases in the aorta, the crural arteries, the arteries of the brain, the inferior cava, the iliac veins, and the coronary veins of the heart.* It has been suggested that, in some cases of sudden death after delivery, the cause might be found in an introduction of air into the circulation by the open mouths of the veins, when the uterus contracted imperfectly. Simpson did in fact find air in such a case, in the uterine veins, the internal iliac, and the vena cava. Another question is the possibility of the spontaneous evolution of gas within the veins during life. Many of the cases on record are undoubtedly mere instances of rapid putrefaction; but we are justified, both by the constitution of healthy blood, and by post-mortem observation, in admitting the possibility of such a change before death. Numerous authors, among whom we would mention Dr. Baillie,† have met with air in the veins of the pia mater in cases of apoplexy, before any traces of decomposition were to be perceived. Dr. Bright ‡ attributes the presence of air in these cases exclusively to accidental injury of the veins, or to incipient putrefaction. The evolution of gas during life, though difficult of absolute proof, is entirely within the range of probability, when we consider that venous blood contains an excess of carbonic acid gas, which is discharged on reducing the atmospheric pressure, as demonstrated by Magnus. "Perhaps," as Professor Puchelt remarks, "it happens more frequently than we are aware, that a bubble of air forms in the venous blood, and again disappears. I am acquainted with at least one variety of palpitation, which produces the sensation, and, I am almost inclined to assert, the noise, as if a bubble passed through a fluid. It occurs generally with but one beat of the heart, and I have met with it in venous subjects with an hæmorrhoidal tendency, and a liability to flatulency."

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* "Medico-Chirurgical Transactions," vol. xvi. p. 418.

† "The History of a Case in which Animals were found in Blood drawn from the Veins of a Boy." London, 1833.

‡ "Gazette Médicale de Paris," 1842, No. 49.

§ Magendie: "Journal de Physiologie," vol. iii. p. 69.

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* See "Puchelt, das Venensystem," vol. iii. p. 328. Liefing, 1843.
† "Morbid Anatomy," p. 430.
‡ "Medical Reports," vol. ii. p. 668.

CHAPTER XXVII.

MORBID ANATOMY OF THE CAPILLARIES.

THE extreme simplicity of the structure of capillary blood-vessels gives little opportunity for morphological changes. We have now to regard them as tubes formed by the union of epithelial plates, each containing a nucleus; but it must also not be forgotten that they are included in a sheath prolonged from the adventitia of the arteries, which is obvious in the brain, and the existence of which is a matter of inference in other parts. The space between the wall proper and the sheath corresponds to the perivascular or lymphatic space round small arteries, and may be called by the same name. We have then to consider variations in the wall proper, in the outer sheath, and in the size or contents of the space between.

It is impossible entirely to separate the morbid conditions of the smaller arteries and veins from those of capillaries, so that some of the following statements will apply also to the two former classes of vessels.

Morbid Changes in the Capillary Wall.—There are doubtless changes in the properties or quality of the capillary wall which are not recognizable under the microscope, and known only by their effects. Thus in purpura hæmorrhagica, there is evidently some change in the vascular wall favouring hæmorrhage, but what that change is we can only surmise. Inflammation produces important alterations in the properties of the capillary wall, which in an inflamed part permits exudation of an increased quantity of serum and of blood corpuscles. It is, however, still uncertain precisely in what this change consists. All that can be usually seen is dilatation, but Cornil and Ranvier state that they have traced also swelling and granular change in the epithelial cells. Cohnheim attributes the capillary phenomena of inflammation to the enlargement of stomata naturally present in the wall. Burdon Sanderson "is led to infer that the primary change consists in the transition of the material composing the vascular wall from the formed into the plastic condition."

Dilatation in chronic inflammation becomes permanent, and is often unequal, so that the capillaries become varicose or irregular in outline. These changes may be traced in the mucous membrane of the bronchi, or in the conjunctiva.

Degeneration of the capillary wall occurs in several forms; the fatty is the most common. Fatty degeneration may be traced in the brain, especially in cases of softening, but to some extent as a normal condition in old age. It is also very obvious in the kidney in some forms of Bright's disease, and there affects both the reticulated intertubular capillaries, and those of the Malpighian glomeruli. The protoplasma of the epithelial plates becomes dotted with fatty granules, sometimes arranged around the nucleus.

Lardaceous or amyloid degeneration is sometimes seen to affect the capillaries of an organ when the disease is in an early stage; the wall becomes thicker, more refracting, and all distinction of structure is lost, so that the tube appears generally more glassy or hyaline. This also is best seen in the kidney. Pigmentary degeneration, if it occurs, results from hæmorrhage.

Changes in the Perivascular Space.—The capacity of this space varies inversely as the state of fulness of the vessels. Thus in the brain Golgi * found that the perivascular spaces were large in anæmic conditions, small in hyperæmia, especially when this condition came on suddenly. Normally this space contains nothing but lymph; it may, however, come to contain blood corpuscles from hæmorrhage, or yellow granules, the result of previous hæmorrhage.

Morbid Changes in the Outer Sheath.—This sheath in capillaries probably participates in the fibroid thickening, often seen in the corresponding parts of the smaller arteries: but the subject has received little attention; a peculiar hyaline thickening has been seen round capillaries of the brain in cases of insanity.

Aneurismal Dilatation of Capillaries.—These vessels may, like arteries, be dilated into minute aneurisms. The vessel is sometimes obliterated beyond the aneurism, which makes the aneurism appear like a terminal dilatation.

Capillary Embolism and Thrombosis.—The obstruction of capillaries, by finely divided matters which are too small to be arrested in the arteries, has been observed clinically, and produced experimentally. Some authors, as Feltz,† would even refer to this cause the production of infarctions, which we have attributed to arterial obstruction. The obstruction of pulmonary capillaries by finely divided fatty matters was first observed by Wagner, who traced the fat to the marrow of bones. We have observed the obstruction of capillaries in the lung by masses looking like agglomerations of leucocytes. Similar obstructions of systemic capillaries have been observed in various parts of the body in cases of pyæmia. Frerichs has supposed obstructions of the same kind to be caused by granules of pigment in cases of ague. Bastian has described a case of obstruction of cerebral capillaries by protoplasmic masses, believed by him to be spontaneously formed in the blood; and attributes delirium to this cause.

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* See "Quarterly Journal of Microscopical Science," vol. xi. new series, 1871, p. 297; Virchow's "Archiv," vol. li. p. 568.

† "Traité des Embolies Capillaires," 2nd edition. Paris, 1870.

The results of capillary embolism will be trifling and temporary, if only a small area is affected; but if the part affected be considerable, necrosis will result. If the material be of such a nature as to injure the vascular wall, hæmorrhage may occur; if it be of an irritating nature, inflammation and abscess. All these results have been produced experimentally.

Teleangiectasis.—Among the chronic forms of disease which are attributed more particularly to the capillaries, and to which we have not had occasion to allude elsewhere, is the affection which Mr. John Bell and English writers following him, have termed aneurism by anastomosis, or the Germans, more classically, teleangiectasis.* It is also known by the simple term, erectile tumour, which is probably the best, as it implies no theory. The affection is commonly congenital, and presents itself in the shape of a cutaneous swelling of a circumscribed form, and bluish-red colour, liable to considerable variations of distension, according to the state of the circulation. The tumour commonly, though not always, offers a pulsation to the touch isochronous with the arterial pulse. Bell described the tumour as consisting of a congeries of vessels, between which were cavities and cells communicating with the latter; others have attributed the affection solely to a distended condition of the vessels, among whom may be mentioned Syme and Pelletan.

Rindfleisch points out that there is not only enlargement of capillaries, but a very abundant new formation of these vessels. It is a vascular hyperplasia, the production of new vessels taking place on his view, by the transformation of connective tissue corpuscles. There appear to be in fact two forms of these tumours, one consisting of vessels only—the simple angiomata; another of vessels with cavities or blood spaces, the cavernous angiomata. The common nævus is the type of the first; the more deeply seated vascular tumours formerly described (p. 149) the type of the second.†

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* Teleangiectasis, literally expansion of the remote vessels, τῆλε, distant, remote, ἄγγεῖον, a vessel, ἐκτεινω, I distend. See also page 168.

† See Lücke: "Geschwülste;" Pitha und Billroth's "Chirurgie," vol. ii. div. i. p. 252; Virchow: "Krank. Geschwülste," vol. iii. p. 307.

CHAPTER XXVIII.

THE LYMPHATIC SYSTEM.

We have here to consider the morbid conditions of the lymphatic vessels and lymphatic glands.

I.—THE LYMPHATIC VESSELS.

Inflammation.—Acute inflammation of a lymphatic is manifested by redness, painfulness, and swelling in its course; the coats become thickened and infiltrated, the cavity dilated, and exudation and suppuration may occur in their channels. A resolution is the most common termination of the process. The presence of actual pus in the lymphatics does not usually arise from inflammation of the vessels; but this may be introduced into them by abrasion or ulceration of lymphatics communicating with an abscess. Puriform matter is, however, frequently found, which, like the softened thrombi in veins, consists of fibrin which has undergone a peculiar molecular disintegration. Suppurative inflammation gives rise to small isolated abscesses along the course of the lymphatic vessels, forming, as it were, stations of the disease, each of which appears to serve as a fresh focus of morbid action. The inflammatory process affects, more or less, the surrounding cellular tissue from the commencement. As the inflammation advances, the lymphatics become blended with it, and suppuration and the formation of abscess involve the entire mass. Inflammation of lymphatic vessels is never primary, but always consequent on some primary inflammation of the surrounding connective tissue. It is seen to be very intense accompanying metritis after child-birth, forming one kind of puerperal fever; and also in the cutaneous lymphatics, in some cases of abscess, poisoned wounds, &c., in the limbs.

Chronic affections of lymphatic vessels are met with in scrofulous, tubercular, and cancerous disease. Their coats are found indurated and thickened, and their channels are blocked up with the heterologous growth. The frequency with which this occurs appears to be in a ratio to the softened condition of the deposit in the organ from which the affected lymphatics take their origin. Thus, in a case of encephaloid cancer of the stomach in an aged female, that occurred under our own observation, the plexus of lymphatics occupying the lesser curvature of the organ was gorged to the size of crows' quills with the cancerous matter.* In the majority of instances, we meet with no such filling up of those vessels, and the secondary affections which are set up in their glands appear to be either the result of some material transmitted to them from the primary seat of the lesion, or of idiopathic disease set up in them by some general cause. Sir Astley Cooper† reports three cases of obliteration of the thoracic duct, two of which were connected with tubercular, the third with cancerous disease. In the first, the obstruction was produced by the thickened valves, in three distinct parts, adhering to one another, the lowest still allowing of a partial transmission of fluid, the upper arresting it entirely. Scrofulous matter was found deposited between the laminæ of the valves. In the second there was considerable thickening and ulceration of the duct; two fungous growths occupied the channel; and in the third, which occurred in a man who had died in consequence of malignant disease of the testis and the lumbar glands, the thoracic duct was found much thickened, and filled with a pulpy mass, composed of broken, coagulable lymph. Opposite the curvature of the aorta the vessel was lost in a swelling as large as a moderate-sized walnut, beyond which it was normal. The paper from which these cases are derived, contain various experiments upon the thoracic duct in animals, of physiological interest. One of the main conclusions arrived at by the author in reference to this point is, that the circulation in the lymphatic system may be kept up by dilatation of collateral vessels subsequent to the occurrence of obstruction in a trunk, as in the vascular system at large.

Dilatation.—A varicose condition of lymphatics is occasionally met with in atonic habits, causing them to resemble hydatid tumours. It affects various parts of the system, and especially the thoracic duct is liable to dilatation; occasionally the entire system is found in this condition, and an extreme instance of this, occurring in a young man, aged nineteen, which happened in the practice of M. Amussat, is given in the works of Breschet and Carswell. Cruikshank‡ alludes to and delineates a similar case. Here the lymphatics of the groins had reached a size sufficient to permit of the introduction of a straw by which air was blown into them; the iliac ganglia had entirely disappeared, and were replaced by the lymphatic vessels. A corresponding enlargement of these vessels was traced through the abdomen into the thorax; none of the other viscera presented any marked pathological changes.

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* "Report of Pathological Society," 1847–48, p. 195.

† "Medical Records and Researches," 1798, p. 87.

‡ See Carswell, "Patholog. Anat. Fasc." ix. pl. iv. fig. 4, and Breschet, "Le Système Lymphatique," 1836, p. 269.

Tissue formed of dilated lymphatics and lymphatic spaces is also seen in the peculiar form of enlargement of the tongue, called macroglossia.

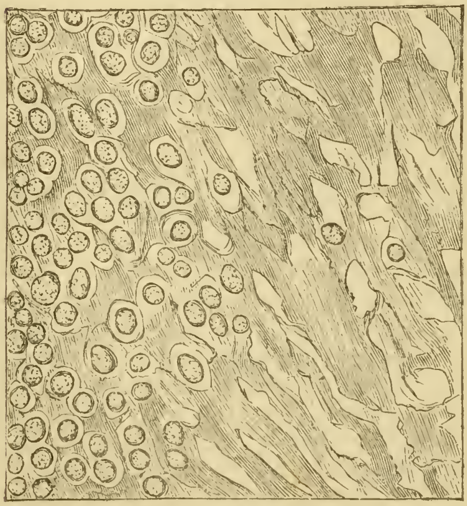
Tumours composed of lymphatic vessels corresponding to the teleangiectasis of blood capillaries have received the name "lymphangiectasis" or "erectile lymphatic tumour" of Anger; while a true cavernous tumour, produced by an actual new growth of lymphatic vessels and spaces, has been described by Billroth and Virchow as lymphangioma cavernosum.*

II.—THE LYMPHATIC GLANDS.

Affections of the lymphatic glands are in most cases secondary, being produced by primary disease in the part with which the glands are connected. The irritation to which their affections are traceable, may proceed from some local lesions, from which it is carried to the neighbouring glands by the connecting lymphatic, as in the case of a sore on the prepuce, inducing bubo, or of porrigo of the head, causing tumefaction of the cervical glands; or it may be excited directly by the morbid condition of the blood circulating in the capillaries of the glands.

Simple acute inflammation is manifested by tumefaction, softening, and a highly vascularized state of the organ, causing it, on division with the knife, to distil blood, while its colour is changed from a reddish grey to a dark red or crimson tint. If suppuration has ensued, yellow spots first appear in different parts, and eventually the entire gland may be destroyed by the process, and be converted into an abscess, in which the surrounding cellular tissue is more or less implicated. The chronic form is, however, the more common; a species of interstitial growth takes place, and induces gradual enlargement and induration of the gland. On the other hand, a reabsorption of the interstitial deposit may occur, as we have frequent opportunities of observing, as the effect of a suitable dietetic and medicinal

Fig. 95.



Cytogenous tissue, with thickened stroma. (Dickinson, "Trans. Path. Soc.," vol. xxi. 1870.)

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* Virchow: "Krank. Geschwülste," vol. iii. p. 490; Lücke: "Geschwülste," p. 267.

regimen, in consequence of which the parts regain their normal size and appearance. The histological changes in the chronic form appear to consist chiefly in the increase of the intercellular fibrous structure or matrix of the gland which may come to predominate over the cellular elements, though normally the converse is the case (Fig. 95).

Hypertrophy of the glands is met with chiefly in childhood, and is often the sign of some constitutional weakness or diathesis, though sometimes no such predisposition can be traced. Enlarged glands may be perfectly normal in structure, and thus be truly hypertrophied, but, perhaps, in most cases the enlargement is complicated either with chronic inflammation or with some of the changes now to be enumerated.

CASEOUS, TUBERCULAR, AND SCROFULOUS GLANDS.

It sometimes happens, especially in the young, that glands which are chronically enlarged, with or without previous inflammation of the parts with which they are connected, undergo a peculiar form of degeneration or metamorphosis, which results in the production of the yellow caseous matter called "yellow tubercle," or "scrofulous matter," which we have already described in the first part. This change occurs especially in persons having what is called a scrofulous constitution, a term which we have before discussed.* Changes very similar are seen in the glands connected with organs in which there is a production of tubercle, using this word in the strict sense formerly explained. When this change is far advanced, no difference can be seen between the cases affected with tubercle and those in which there is no disease or simple inflammation of the adjacent organs. Hence all these changes were at one time confounded together under the same name. Subsequently, by examining early stages of the metamorphosis, pathologists (especially Virchow) have shown that the strictly tubercular disease differs anatomically as well as clinically from that in which no tubercles are present.

We have accordingly to consider separately the scrofulous and the tubercular affections of the glands.

Scrofulous Glands.—The changes in lymphatic glands, called scrofulous, appear to be always dependent on some morbid changes in the organs whence the glands derive their lymph, and especially on inflammatory changes. There is, however, this peculiarity, that the primary or exciting cause may be acute and transitory, and the resulting secondary affection of the glands chronic or permanent.

The morbid process in the glands consists essentially in multiplication of the cellular parts or lymph corpuscles, and hence is at first a simple cellular hyperplasia. The newly-formed lymph cells have, however, certain peculiarities; they are imperfectly organized, and have little permanence; they soon undergo an imperfect fatty metamorphosis, which either leads to complete absorption and resolution, or else to a merely necrobiotic change, the perished cells remaining mingled in one mass with the new ones which are continually formed.

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* See p. 91.

The gland in its early or hyperplastic stage has a uniform pale grey, translucent appearance, though sometimes more opaque, and either white or pinkish. Sometimes it is more succulent than a healthy gland, but in general drier and denser, though the vessels are prominent and full.

This condition does not last long; the gland becomes denser and tougher, and the necrobiotic decay of the newly formed elements begins; yellow patches begin to appear in the grey mass, and finally the whole gland may be converted into a firm, tough yellowish opaque mass, the appearance of which has been compared to that of a raw potato, only that there is less moisture. This is called the caseous metamorphosis, but in fact the dry crumbling consistency which suggested this name is only gradually developed out of the condition just described.

The caseous mass is still susceptible of further transformations. Sometimes softening takes place, by a kind of imperfect solution, so that a turbid puriform fluid results, containing only *débris* of tissue, fatty molecules, and the like, which has however been called scrofulous pus. This may open externally, forming a scrofulous ulcer, and the morbid material be completely eliminated, so that only the well-known scrofulous scar remains. Again, another change is possible, consisting in more complete drying up and deposition of lime salts, so that either an atheromatous paste containing cholesterine and other fatty products, or else an actual calcareous mass, results.

Complete resolution and absorption are also even at this stage possible. Actual suppuration is not common at any stage of the process above described, but may under some circumstances take place.

Children are peculiarly prone to scrofulous and tubercular disease of the glandular system; but there is a considerable difference in the proclivity of different sets of glands to be affected. All authors are agreed that the bronchial are pre-eminently endowed with this tendency. The analysis of one hundred post-mortem examinations of children affected with scrofulous and tuberculous disease, by Dr. Lombard,* showed that

The bronchial glands	were	affected in	87	cases
The mesenteric	do.	do.	31	,,
The cervical	do.	do.	7	,,
The inguinal	do.	do.	3	,,

In the case of the bronchial glands a communication is occasionally established between their contents after they have softened and the channel of the bronchi, and may be thus evacuated by expectoration. The chalky concretions find their way out occasionally in the same manner. A remarkable instance of death being caused by the impaction of the cretaceous contents of a bronchial gland in the bronchi, is detailed by Dr. Tice in the twenty-sixth volume of the "Medico-Chirurgical Transactions."

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* Andral: "Précis d'Anatomie Pathologique," vol. i. p. 425.

Caseous glands are very frequently found in the bodies of persons dying of phthisis, and hence it has been thought that tuberculosis is a secondary disease resulting from the absorption of this caseous substance, which is more often seen in glands than elsewhere. The editor of this work observed a case where, by subsequent softening of a scrofulous gland, some of this material was introduced into the superior vena cava; producing, first, local coagulation and softening of the thrombus; secondly, general pyæmia and suppuration in the joints.

Tubercular Glands.—Changes due to the production of tubercle in the glands are seen both in the acute and chronic condition. The former occur in cases of acute tuberculosis. The glands are then found highly vascular, succulent, and more or less enlarged, while greyish miliary tubercles may sometimes be seen in them. More often we find a marbled or mottled appearance, parts of the gland being opaque and white, while the remainder is vascular. The same whiteness and opacity may extend over the whole gland, and be succeeded by dry necrosis or caseation; but another change is also possible, and according to Virchow, frequent; which consists in induration and production of fibroid tissue, accompanied by thickening of the capsule, and adhesive inflammation of the surrounding tissue; the caseous change being then limited to isolated spots. Ulceration, like that of scrofulous glands, is, however, possible, and so is calcification; a formation of peculiar concentric calcareous corpuscles has also been observed by Virchow and Schüppel.

The histology of tubercular glands is a subject on which some difference of opinion exists. It is described by Virchow * somewhat as follows. The production of tubercles takes place in the fibrous septa of the gland, not in the follicles, the latter parts being unaffected, or simply hyperplastic, and it thus contrasts with the changes in scrofulous glands which are chiefly in the follicles. The tubercles are of what Virchow has distinguished as the fibrous variety; they contain few round lymphoid cells, but are on the contrary made up of cells resembling epithelium and large many-nucleated or myeloid cells, so as even to recall a myeloid tumour. Beside this there is much general proliferation of the connective tissue, which does not necesarily follow, but may even precede the development of the tubercle.

More recently Schüppel † has given a very different account of the same matter. According to him the tubercles are always formed in the follicles of the gland, and have nothing to do with the septa, or the lymph paths. The commencement of each tubercle is in a many-nucleated, myeloid, or "giant" cell, which may be complex in form, with many branches, and containing even as many as fifty or sixty nuclei. This original myeloid cell takes its rise in a vein or some small blood vessel, and is evolved from structureless protoplasm. This last explanation he would extend to all tubercles. Retrogressive changes soon set in, the life of the tubercle being very short; and it either becomes simply necrotic or else undergoes a fibrous transformation. With respect to the character of the cells he agrees with Virchow.

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* "Krankh. Geschwülste," vol. ii. p. 672.

† "Untersuchungen über Lymphdrüsen-tuberculose." Tübingen, 1871, etc.; see "London Medical Record," vol. i., Nos. 3 and 4, 1873.

Tubercle of the lymph glands is generally secondary to tubercular disease of the organs connected with the gland; but this is not always the case, for those organs may have some disease unaccompanied by the production of tubercle, viz., either degenerative and destructive ("scrofulous"), or simple inflammation. These must be called cases of primary tuberculosis of the gland, and may be compared to those cases of tubercle of the pleura following non-tubercular disease of the lung.

Schüppel contends that all caseous or scrofulous glands are originally tubercular, and that the caseous process is nothing but necrosis of tubercle. He thinks that in a large number of cases the tubercle in the glands is the primary tubercle, and that its formation is the result of local irritation combined with constitutional predisposition.

Syphilitic Affections of Lymphatic Glands.—In syphilis the lymphatic glands undergo a sort of chronic inflammation which has some resemblance to that called scrofulous. The glands enlarge, but not usually to any great extent; they become hard and suffer more fibroid change than is seen in the scrofulous process, while only partial caseation or yellow necrosis takes place. These changes begin in the glands which receive their lymph from parts affected with the primary syphilitic lesion; but secondary affections may also excite similar changes in the glands, and when once commenced, the process may extend to distant glands. Acute inflammation and suppuration often occur in glands dependent on parts where there exists a suppurative or soft chancre, and are not in themselves syphilitic; but the occurrence of suppurative glands or buboes does not exclude syphilis.

Lardaceous Degeneration of Lymphatic Glands.—This change occurs in the glands when other organs are similarly affected, but probably never alone, and not at all commonly. The appearances are similar to those seen in other parts affected with the same degeneration.

Changes in the Glands from Enteric Fever.—In this disease the morbid changes in the intestines are accompanied by similar changes in the mesenteric glands. When we find in the ileum swelling and ulceration of the Peyer's patches, the mesenteric glands immediately dependent upon that portion of the bowel are found enlarged, hyperæmic, and infiltrated with a creamy juice containing abundant lymph corpuscles. The change, like that in the intestinal glandular structures, is a lymphatic hyperplasia characterized by a profuse production of cells, as was long ago pointed out by John Goodsir. The newly-formed cells show, however, a marked tendency to degeneration and necrosis, which assimilates the process in some degree to scrofulous changes; actual softening and suppuration do not occur, but yellow necrotic masses may be produced.

Leuchæmic Enlargement of the Glands.—In the disease called leuchæmia, enlargement of some of the lymphatic glands is common if not constant. (See p. 74.) The enlargement presents generally the character of a pure hypertrophy, but in some cases, according to Virchow, the development may become heteroplastic, and the growth spread beyond the glands into the neighbouring tissues. Another way of stating this is to say that leuchæmia sometimes accompanies the growth of infiltrating or infective lymphoma, and sometimes not. The extension is probably to be attributed to the migratory properties of leucocytes. These hypertrophies thus approximate to new growths proper, which we have now to consider.

NEW GROWTHS OF LYMPHATIC GLANDS.

Primary new growths of the lymphatic glands are far less common than secondary, the latter being dependent upon similar disease either in the part from which the gland derives its lymph supply, or in some distant organ.

Primary new growths are chiefly lymphoma and sarcoma. Carcinoma in the form of encephaloid has also been often described, and in some two or three cases epithelioma is said to have occurred as a primary growth.

Lymphoma or Lymphadenoma.—This growth begins with simple enlargement or hyperplasia of the gland, and we cannot give any precise definition to separate the tumours from simple enlargements. The latter term becomes, however, inappropriate when the structure becomes different from that of a normal gland. The difference may consist in a greater development of the intercellular stroma leading to induration, and a more homogeneous, elastic, translucent consistency in the gland, or in a preponderating development of cells, so that the tumour appears to be entirely cellular.

The specially cellular forms are called by Virchow, in accordance with his system, lymphosarcomata. They are often infective and infiltrate neighbouring parts, or are, in other words, malignant. He distinguishes a soft and a hard form, but we find it impossible to separate the latter from other forms of enlargement. The former are a more distinct class.

Soft, or Medullary Lymphoma—Lymphosarcoma.—A good type of this form of tumour is seen in the soft rapidly-growing tumours of the mediastinum. These originate usually in the bronchial glands, though in some cases the starting point has been thought to be the thymus. In an early stage they appear like simple enlargements of the glands, but as they increase, the growth extends to the surrounding tissue by a species of infiltration. In this way not only the whole of the mediastinal connective tissue, but adjacent portions of the pleuræ, pericardium, and the thoracic walls will be involved in a lymphatic new growth, and very large masses are thus produced. In other cases the lung tissue is invaded, or even the heart, and the large veins, are penetrated by the new growth. These tumours possess a structure closely resembling that of a lymphatic gland, namely, a great abundance of lymphoid cells enclosed in a finely-reticulated stroma. The latter sometimes assumes an alveolar structure like that of carcinoma, and, in fact, such tumours resemble cancer in everything but the "epithelial" character of the cells. They make up the greater part of what has been described as intra-thoracic cancer, corresponding precisely in their naked eye characters to what was formerly taken as typical medullary cancer, or encephaloid.

These tumours have accordingly most of what are called malignant properties; but they rarely give rise to secondary growths in distant parts, and when such growths occur simultaneously in other organs, such as the spleen, this must, in some cases, be regarded as the hyperplasia of similar tissue rather than as a secondary production.

Soft lymphomata of the same kind and magnitude as those occurring in the mediastinum are less frequently developed in other glands, except in the cervical glands; these sometimes give origin to large tumours, which may infiltrate the neighbouring parts, and enter the thorax. The retroperitoneal glands of the abdomen are also sometimes the starting-point of large lymphosarcomata. In most of the other glands, tumours if formed are of the hard kind.

Hard Lymphoma.—Although such a character as hardness cannot of course be generally regarded as of much importance, it serves in the present instance to distinguish from the medullary, highly infective, and malignant forms just described, others in some respects similar, which do not infiltrate the neighbouring parts, or possess in general such malignant properties. They are described by Virchow as showing proliferation of the septa, and production of cells from all parts of the gland, with much increase of connective tissue; but it is doubtful whether they can really be distinguished from hypertrophy, at least in the early stages. Later on the connective-tissue increase causes the cells to become fewer, and there is usually thickening of the capsule. These tumours occur especially in the neck and axilla; they never attain any great size; being seldom larger than a plum or an egg.

Anæmia Lymphatica; Hodgkin's Disease.—Cases are sometimes met with in which there is an enlargement of many lymphatic glands throughout the body, and at the same time masses of new growth resembling lymphatic tissue are formed in internal organs, especially the spleen. These changes have been supposed to indicate a special disease analogous to, but not identical with leuchæmia (since there is no increase in the white corpuscles of the blood); but we are here only concerned with the changes in the lymphatic glands.

These have been described in very different terms by different observers, the variations depending apparently upon the stage of development at which they happened to be examined, as well as upon the preponderance or subordination of cell-growth. Moreover, it is not certain that really different processes may not, as producing similar symptoms, be included under the same name.

When different stages of development are met with in the same body we find the earlier forms (in the smaller glands) much more vascular and softer than the more advanced, which are pale, tough, and elastic, and often almost homogeneous and translucent, or even waxy in appearance. The minute characters correspond to these external differences. We find in the smaller forms a great abundance of cells, and these not always like ordinary lymphoid cells, but often larger and irregular in shape; while myeloid or giant cells are not unfrequently seen, as in other hyperplastic conditions of the lymph glands. When the gland increases in size the cells multiply, but are individually smaller, and abundant nuclei are produced. In this stage the structure has been described as "fibro-nucleated." In more advanced stages the stroma increases, while the cells diminish relatively, and probably actually waste. The bands of the stroma at the same time increase greatly in width, till the appearance characteristic of chronic inflammation of the glands or adenoid tissue with preponderance of intercellular substance is produced (see Fig. 95.) The latter change may ultimately reach so extreme a degree that the whole substance appears even under the microscope almost homogeneous, and much resembles a part affected with lardaceous degeneration, though iodine produces no change. This condition has hence been described as one produced by albuminous exudation. It is not known that any further transformation takes place, since the glands do not become affected with caseous degeneration, softening, or calcification, like scrofulous glands; only some slight approach to this condition being occasionally observed. No peculiarity of chemical constitution is known to exist.

Sarcoma.—Beside the tumours called lymphosarcoma, already described, new growths may arise in the glands which have the characters of true sarcoma, either round-celled, or spindle-celled. The retroperitoneal glands, near the head of the pancreas, are not unfrequently the seat of a growth of this kind, which often secondarily affects the liver. Other similar growths occur in the cervical and axillary glands, which may equally give rise to infiltration of the surrounding parts, and then be reproduced in distant organs; sometimes by embolism, as the growths often perforate the walls of veins.

Secondary growths of sarcoma also often occur, as in the inguinal or femoral glands, subsequently to the occurrence of a similar tumour in the limb. This secondary infection is not quite so common as in carcinoma.

Carcinoma.—Taking this designation in the strict sense in which it is now understood it appears doubtful whether it ever occurs primarily in lymphatic glands. As a secondary growth it is, however, extremely common; and the usual path by which a primary carcinoma infects the whole body is by infecting first the lymphatic glands.

Epithelioma.—A very few cases have been described of this growth occurring primarily in lymphatic glands. Sir J. Paget described some years ago such an affection of the inguinal lymphatic glands on both sides in a chimney sweep. Förster speaks of a similar case; and a few more are recorded by Langenbeck as occurring in the neck; the latter are supposed by Lücke* to have grown from subcutaneous dermoid cysts. Secondary affections are extremely common.

MELANOSIS.

An affection to which the glandular system, and especially those parts in relation to the respiratory organs, is very prone, is melanosis; it is not, however, to be looked upon as a morbid process in itself, but rather as an evidence of the depurating functions by which they assist in eliminating the superfluous carbon from the body. We have shown elsewhere that melanosis does not, in itself, constitute a malignant disease, and that it does not consist of a new formation, though frequently complicated with some new growth. While it is unusual to find black matter in the lymphatic ganglia of the abdominal or inguinal regions, we constantly meet with it in the glands surrounding the bronchi. Much of the pigment here seen is undoubtedly carbonaceous matter inhaled with the breath and conveyed from the lung by lymphatic vessels. Another part, again, is due to hæmorrhage in the lung.

ENTOZOA.

Almost the only known instance of entozoa being found in the glands, is recorded by Rudolphi.† It was discovered by Treutler, in a person worn out by syphilis; it was an inch in length, tawny in colour, semi-transparent at one end, presenting two hooklets at its anterior extremity, and hence termed hamularia bronchialis. Förster refers to a still earlier observation by Bianchi, of thread-worms in the bronchial glands.

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* Pitha und Billroth: "Chirurgie," ii. 1, p. 207.

† "Entozoorum Historia Naturalis," vol. ii. p. 82.

THE PATHOLOGICAL ANATOMY OF THE ORGANS OF RESPIRATION.

CHAPTER XXIX.

GENERAL OBSERVATIONS.

WE shall examine the pathological conditions of the different parts of the respiratory system in the order in which they naturally present themselves, as we proceed from the orifice downwards; commencing with the larynx and its appendix the epiglottis, we shall descend to the trachea, the bronchi and their ramifications, the pulmonary parenchyma and the pleura. Though a definite relation exists between these different parts, which is more intimate in some than in others, they are each susceptible of isolated morbid states; and though the continuity of the mucous membrane lining the entire passages frequently induces a propagation of disease from one part to the other, this is by no means universally the case, and the catarrh or croup affecting the larynx or trachea need not cause any pathological changes in the adjacent mucous membrane, or the other tissues of the affected part itself. The absolute importance of the morbid condition to the individual does not necessarily bear a direct relation to its intensity, but depends a great deal upon the part affected; a point which the physiological laws regulating the different sections of the respiratory organs render sufficiently palpable; thus a trifling amount of inflammatory swelling of the glottis or epiglottis threatens danger, while much more intense inflammation of the pulmonary parenchyma, or of the bronchi, may run its course without causing more than a temporary inconvenience. The features of the morbid condition may be identical, while their bearings upon the existence of the individual are widely dissimilar.

INFLAMMATION OF MUCOUS SURFACES IN GENERAL.

Before entering on the diseases of the respiratory mucous membrane, we shall endeavour to classify the forms of inflammation which can affect such surfaces, since they present so close a general agreement that much special description will thus be saved. The following account applies not only to the respiratory mucous tract, but, with some modifications, to the intestinal, and in a more restricted sense, to the genito-urinary tract, and to the eye and ear.

Three forms of inflammation are distinguished which may affect mucous surfaces :—

1. *Catarrhal inflammation*, the form peculiar to such surfaces, in which no solid adventitious products are formed.
2. *Croupous inflammation*, in which solid sheets called false membranes, are formed upon the diseased surface. This has more resemblance to the inflammations of serous membranes.
3. *Diphtheritic inflammation*, in which there is necrosis or sloughing of the tissues, with the formation of false membranes much resembling those of croupous inflammation.

The two latter forms are comparatively rare ; the former very common, being the almost invariable result of any injury or irritation of the mucous tissue.

I.—CATARRHAL INFLAMMATION.

In catarrhal, as in other inflammations, we have *hyperæmia*, shown by the more or less pronounced red colour ; *hyperæsthesia*, shown by the extreme sensibility of the mucous surface ; *increased productiveness*, shown in the swelling from interstitial effusion, in the abundant liquid secretion (flux or catarrh proper), and in the numerous leucocytes or *young cells* which the secretion contains. There is also *increased waste* or destruction, shown by the detachment of very many epithelial cells, by the fatty degeneration of many of these, and by the molecules of fat present in the secretion. It thus presents all the essential characters of inflammation as formerly defined. The factors of the process must now be separately considered.

Hyperæmia is doubtless the most important condition of catarrh, and often must be regarded as the cause of all the other derangements. Venous hyperæmia very commonly produces or predisposes to catarrh, as is seen in the occurrence of bronchitis, in consequence of cardiac obstruction ; of gastric and intestinal catarrhs, from obstruction of the portal system, &c. Whether simple, active, or arterial hyperæmia, such as that resulting from section of the sympathetic can of itself be said to produce catarrh, is doubtful ; but, at all events, it brings the mucous surface into such a condition that very slight causes will set up inflammation. On the other hand, active hyperæmia is an immediate consequence of the morbid stimulus (whatever it may be) which produces catarrh, and is therefore one of its earliest symptoms. Hyperæmia has for its immediate consequences redness, hypersecretion, and swelling ; it may lead to increased temperature; and doubtless has a share in the production of the hyperæsthesia of inflamed mucous surfaces.

The redness of a catarrhal mucous membrane varies much in tint and intensity. In early stages it is a bright red arterial colour, but when catarrh is connected, as it often is, with *venous* congestion, the tint is more nearly purple; and the latter is also more characteristic of chronic catarrh. In the acute stage, numerous small hæmorrhages heighten the red colour; and through the gradual changes, undergone by this blood pigment, we get the slaty colour or pigmentation which long-continued catarrh always produces. It is most important to remember that the redness due to mere turgescence of the vessels in the acute stage may be quite unrecognizable after death, the vessels having emptied themselves by their elasticity.

Textural Changes.—Swelling of the mucous membrane is a very constant phenomenon: it appears to depend on several causes beside the turgescence of the vessels. These are as follows:—Enlargement of the individual elements in consequence of more active nutrition; interstitial effusion of serum which infiltrates the parts; and the production of new elements. These new elements, or young cells, are of the kind usually met with as inflammatory products, being in fact leucocytes, undistinguishable from those of the blood, and showing amœboid movements. As in other inflammations, some doubt still hangs over the origin of these elements, and it is uncertain whether they are migratory cells derived from the blood, or whether they are produced by proliferation of the tissue elements, viz., of the sub-mucous connective tissue, or of the epithelial cells themselves. With respect to the corpuscles of the sub-mucous connective tissue they show unmistakeably the appearances regarded as indicating cell-multiplication by division. The relations of the epithelial cells are more obscure. Large compound cells, inclosing in their substance smaller young cells, have repeatedly been observed, which Remak, Buhl, and others interpreted as *mother-cells*, propagating, as in the embryo, by "endogenous multiplication." More lately it was shown that large swollen epithelial cells were capable of receiving migratory cells into their substance, and that structures presenting the appearance of endogenous proliferation might thus be produced, and the researches of Bizzozero* seem to have shown conclusively that in certain inflammations, viz., hypopyon or suppuration in the anterior chamber of the eye, this is actually the origin of what have been regarded as compound or "mother-cells." The participation of epithelial elements in the production of young cells in catarrhal inflammation must therefore be regarded as not yet satisfactorily established.

Secretion is not only enormously increased in quantity (for the normal secretion of a mucous surface is extremely small in amount) but also much altered in quality. The mucine is greatly increased, and the water relatively diminished; but the special constituents of the secretion, if any (such as those of the gastric surface), are not increased. The numerous cells floating in the secreted liquid greatly modify its appearance and properties. They are in general more abundant as the mucus is deficient, and thus two chief types of catarrhal inflammation are marked out, the mucous and the purulent. These types may succeed one another in the course of the same disease, or else exhibit their distinctive character from the beginning.

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* Stricker's "Medizinische Jahrbücher," 1872, p. 160.

Ulceration is not a common consequence of catarrh, and occurs, if at all, usually in chronic cases; but it is not necessary to attribute it to any other special cause beside the catarrh itself. The so-called catarrhal ulcers are generally shallow, only superficially eroding the mucous membrane proper. Changes in the submucous tissue (such as induration) sometimes accompany chronic catarrh. The fever accompanying this form of inflammation has some more or less clearly defined characters. Its onset is gradual rather than sudden, often marked by repeated rigors, and the temperature does not reach the same height as in some inflammations. There is, however, a marked tendency to become chronic even in cases which may have had an abrupt beginning; *e.g.*, bronchial catarrh, gonorrhœa, conjunctivitis, aural catarrh, and others.

II.—CROUPOUS INFLAMMATION OF MUCOUS SURFACES.

Although, as we have said, the ordinary forms of inflammation on mucous surfaces do not give rise to any solidifying exudation like the "inflammatory lymph" of some other parts; there is a disease, generally very distinct from ordinary catarrhal inflammation, in which are produced considerable sheets of adventitious matter, usually called false membranes. These have been thought to be composed of fibrin, produced out of materials exuded from the blood-vessels, but there are several difficulties in the way of this view, and of late years it has been contended that the apparent exudation is nothing but the greatly altered tissue itself, shed off from the surface.

Opinions still vary about the true nature of the croupous false membrane, and we can only give the description of it which we believe to be the most accurate, that of Wagner, which has been essentially confirmed by Buhl, Rindfleisch, and others.*

Histology of Croup.—The microscopical investigation of the croupous membrane (according to Wagner) shows a clear homogeneous network, of highly refracting substance; the roundish meshes of which contain serum and pus corpuscles (leucocytes), sometimes epithelial cells, free nuclei or red corpuscles. The network itself has a great power of resistance to heat and chemical reagents. It is at first covered by the topmost layer of epithelium (here ciliated), but later on this disappears. The membrane accordingly replaces the epithelium, and is formed by a peculiar metamorphosis of its cells, which become partly absorbed, partly converted into pseudo-fibrinous substance. The change has been called by Wagner croupous metamorphosis, and is thus described by him. In the first place, a general enlargement of the cell, especially of its protoplasma, takes place. Next appear several round or oval spaces, which ultimately become vacuoles, the cell-substance completely disappearing, while the remaining substance becomes darker, more refracting, and looks jagged in outline. Finally, the cell is quite perforated by these spaces, which may also destroy part of its outline, and the nucleus disappears; but its residue is composed of substance extremely resistent to reagents, and becomes firmly united to the neighbouring cells, so that an open but very firm network is produced. The leucocytes found in the meshes of this structure are probably, like the occasional red corpuscles, derived from the vessels.

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* Wagner: "Archiv der Heilkunde," vol. vii. p. 481, vol. viii. p. 449; Uhle und Wagner: "Allgemeine Pathologie," 5th edition, 1872, pp. 285, 361; "Buhl Verhandl. der Bayr. Acad. der Wissensch," 1863, vol. ii. p. 59; Rindfleisch: "Pathol. Histol." 2nd edition, p. 290; Murchison: "Trans. Path. Soc.," vol. xxii. p. 35, 1871.

The above description gives us the anatomical definition of croup, but includes some forms of disease usually called diphtheria, such as ordinary epidemic "diphtheria" of the pharynx. Clinically distinct as these forms are, an anatomical distinction corresponding to the two types of disease has not yet been found; that is to say, both croup and diphtheria may affect the pharynx as well as the air passages.

III.—DIPHTHERITIC INFLAMMATION.

This kind of inflammation is, like the last, accompanied by the production, on the surface, of apparently adventitious material in the form of layers or false membranes. They greatly resemble croupous false membranes, and though several criteria have been suggested for distinguishing one from the other, it cannot be said that any of these are perfectly satisfactory. The type of diphtheritic inflammation or diphtheria is commonly taken from a disease of the pharynx and tonsils, which occasionally extends to or affects the air passages, while croup (as generally understood) is a disease of the larynx and trachea. But, on the other hand, there are affections of the tonsils and pharynx which some authorities call croup (Wagner and Rindfleisch).

What is strictly called diphtheria is, in a high degree, locally contagious, so that it may not only spread from its original centre, but be transferred accidentally to other parts of the body or to open wounds; its original locality is therefore not all-important. We must state that the term diphtheria is used on the Continent in a much wider and indeed quite different sense from that which has been usual among ourselves; and that an almost inextricable confusion has resulted from the application of the same name to different objects. It is difficult to separate wholly the morbid anatomy from the clinical symptoms which we cannot discuss here; and our exposition must therefore be very imperfect.

Anatomical Characters.—In well-marked cases of diphtheria, a grey or ash-coloured false membrane is formed on the diseased surface, and can only be detached with difficulty, and not without some injury to the subjacent surface, shown either by bleeding or by ragged ulceration. It is in fact a part of the mucous membrane: being partially, at all events, made up of necrotic sloughing tissue. This character of *necrosis* is the only distinguishing one of diphtheria; but it may have, in addition, the characters of croup. Microscopic examination of the false membrane shows chiefly necrotic tissue; at the same time, minute organisms, resembling bacteria, are seen in it, and sometimes a kind of fungoid growth appears on the surface. One or the other of these organisms has been regarded as the cause of the disease; but since they are commonly met with, or likely to occur, on dead and decaying animal tissue, it is possible that they may be rather the result than the occasion of the morbid processes.

THE EPIGLOTTIS.

The epiglottis is, notwithstanding its exposure, not very liable to disease, a circumstance due in part to the dense fibro-cartilaginous tissue which forms its substratum. The mucous membrane which invests it, may be affected with acute or chronic inflammation, in which case the vessels enlarge and become tortuous and congested, and the light pink hue is converted into a streaky, or more or less uniform redness. The acute form is commonly the result of mechanical injury or chemical irritation; while the chronic form accompanies old standing catarrhs of advanced age, arthritic or syphilitic cachexia, and other conditions resulting from general atony. The former is met with as a result of the action of irritant poisons, which may thus cause death without passing the fauces; and as Dr. Marshall Hall* has pointed out, in consequence of children, as frequently happens among the poorer orders, drinking boiling water from the spout of the kettle or tea-pot. In these cases there is also rapid and extensive œdema, one form of the well-known and formidable œdema of the glottis, giving rise to a mechanical impediment to the admission of air into the lungs. The affection consists essentially in a serous infiltration of the submucous tissues producing immense swelling, but not going on to suppuration. In one of the instances detailed by Dr. Davis,† suffocative dyspnœa supervened within three or four hours, and when at this period the attempt was made to apply leeches, the child, a little girl of three years of age, was much terrified, and screamed so violently, that they could not be used. From this moment, however, respiration became easy, and a speedy recovery took place. The remedy which in such a case relieves the immediate symptoms, and may thus be the means of saving life, is the operation of tracheotomy, though, as in the instance from which the delineation was taken, not always successful.

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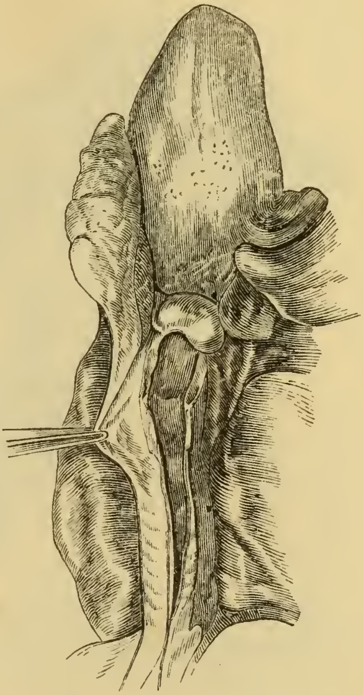
* "Medico-Chirurgical Translations," vol. xii. p. 1.

† Ibid.

Fig. 96.



Œdema of the epiglottis, brought on by drinking scalding water, and causing the death of the child, a boy aged three years and nine months, in two hours. The uvula, which is seen a little above and to the right of the epiglottis, was also œdematous. No other feature was observed, except some reddening of the bronchial mucous membrane, and congestion of the lungs. Tracheotomy was performed.

Œdema of the epiglottis alone, without participation of the neighbouring parts, occurs rarely; and, when it does, is confined to the upper half. Œdema of the lower half is accompanied by the same affection of the upper part of the larynx.*

Ulcerations.—Ulcers of the epiglottis are not unfrequent, more especially as complications of phthisis; they are generally limited to the inferior surface of the valve and to its mucous covering; though they occasionally penetrate to the fibro-cartilage. They are not generally accompanied by much tumefaction or reddening of the surrounding tissue. Louis† states that in all the cases that fell under his notice, he only once observed ulcerations on the lingual surface. He found the breadth of the ulcers to vary from about one to two lines. In some cases the laryngeal surface of the epiglottis was entirely deprived of its mucous membrane: in four the edge of the fibro-cartilage was destroyed, as well as the surface ulcerated, so as to give the part a festooned appearance; in a fifth the epiglottis was totally destroyed. The proportion of phthisical cases in which the epiglottis is affected, appears, according to the same author, to be about one-fourth. These ulcers are not, however, the result of a fusion of tubercular deposit, which seems not to be met with at this point, but of the accompanying inflammation; they, like other affections of the mucous membranes of the air passages which have been mistaken for tubercular deposit, often originate in occlusion and sebaceous enlargement of the numerous solitary follicles scattered about the respiratory mucous membrane. These vary in size from an almost imperceptible point to a pin's head, and even to that of a bean;* and closely resemble miliary granulations, surrounded by a halo of blood-vessels, as they appear for instance in the pia mater. The microscope, however, reveals their structure; showing the basement membrane of the follicle lined with epithelium, and containing oily matter. The cheesy particles coughed up by many people, are formed partly in the follicular structures of the tonsils; and probably also in the follicles of which we have just spoken. The epiglottis is liable to suffer from syphilitic ulceration, by an extension of the disease from the fauces: it very rarely passes beyond to the larynx and trachea. In small-pox and other eruptive fevers, the peculiar poison of the disease often sets up its action in the fauces and the respiratory passages; the former may give rise to pustules on the glottis, accompanied by a more or less redness and serous infiltration; in the latter ulcerative destruction and œdematous swelling occur.

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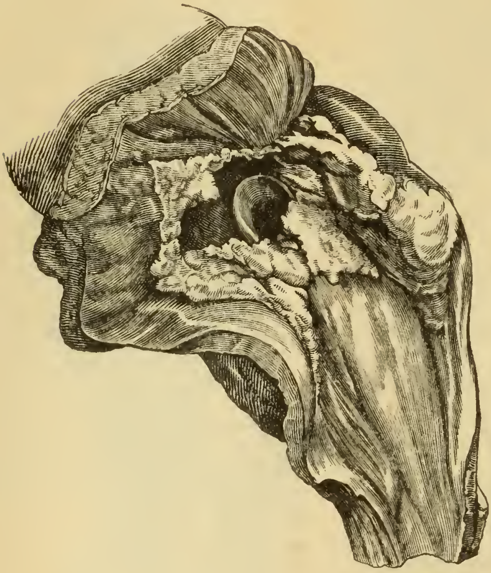
* For a case of acute inflammation confined to the epiglottis see "Trans. Path. Soc.," 1864, vol. xv. p. 50.

† "Researches on Phthisis," Sydenh. Soc. Ed. p. 42.

Fig. 97.



Acute ulceration of the epiglottis and surrounding parts in a man, caused by taking a large dose of bichloride of mercury. Death followed after nine days; there were pneumonia, ulceration of the stomach, and inflammation of the entire intestinal tract.

THE LARYNX.

The mucous membrane of the larynx presents the same changes that we meet with in the mucous membrane investing the entire respiratory tract. In health it has an almost colourless, slightly pink hue, and consists of a uniform epithelial surface, overlying a basement membrane, underneath which the vascular network ramifies. The surface is broken only by the minute follicles which open upon it. In congestive and inflammatory states this vascular network becomes much distended; the membrane is rendered thick and soft by mere repletion in the first instance, and subsequently by interstitial effusion. The more lasting and chronic the inflammatory action, the more this thickened condition is established, and thus a permanent hypertrophy of the membrane is effected. The colour of an inflamed respiratory mucous membrane varies according to the intensity, and also according to the character of the inflammation, from a bright crimson or scarlet, to deep purple or dusky redness. The hyperæmia is often found to be entirely local; this is chiefly the case in chronic affections: thus we may find it limited to the larynx, the trachea or the bronchi. The character of the inflammation necessarily determines the nature of the products which occur upon and beneath the membrane; thus we find the three forms of inflammation characteristic of mucous membranes, the catarrhal, the croupous, and the diphtheritic.

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* See a case detailed in Dr. Bright's "Reports," vol. ii. p. 644.

Catarrhal Inflammation.—The most acute form is probably that in which, from the contact of a powerful irritant, such as boiling water, an instant effusion of serum takes place in the submucous tissue, causing its distension or œdematous swelling. We have already alluded to the cases in which children have met with their death in consequence of drinking the hot contents of a kettle from its spout. In some of these it appears that the œdema rather affects the parts below the epiglottis than the epiglottis itself. Here it is the mechanical effect of the tumefaction of the loose tissue at the glottis and not the intensity of the pathological process, that produces the fatal issue.

In catarrhal inflammation of the larynx, the dyspnœa, and affection of the voice, depend mainly upon the amount of tumefaction at and near the rima glottidis; during the first stage there is, as elsewhere, in the mucous membrane a sense of titillation, roughness, and pain; and as the stage of secretion supervenes, these symptoms subside and are relieved by expectoration. This presents various appearances to the naked eye; at first it is a glairy viscid mucus, which subsequently assumes a greenish or yellowish colour, losing its adhesiveness and forming into opaque rounded pellets. If a blennorrhœic state supervenes, the discharge assumes a purulent character. Under the microscope these varieties can scarcely be distinguished. In each we find mucous corpuscles and epithelium; and if the inflammation has been of sufficient intensity to cause hæmorrhage, we shall also discover blood-corpuscles. Idiopathic laryngitis appears generally to be the result of atmospheric changes; it runs a rapid course, and it is one of those affections in which the performance of tracheotomy may become the means of saving the patient's life. The disease acquires an historical interest, from the circumstance that it proved fatal to Washington. Rokitansky describes a variety which he considers important on account of its leading to a contraction of the passage. He states it to attack the mucous membrane of the epiglottis and the duplicatures of the glottis, converting the mucous membrane and subjacent areolar tissue into a fibro-lardaceous white resistent structure of tolerable thickness, thus giving rise to contraction of the rima glottidis and the cavity of the larynx.

In children, catarrhal inflammation of the larynx may produce all the symptoms of croup; in which case, though, as Andral observes, the passage of air through the contracted rima gives rise to the well-known sound of croup, the post-mortem examination will only exhibit a slight tumefaction of the mucous membrane, without any trace of the membranous exudation, which is characteristic of that disease. Chronic catarrh of the larynx may lead to ulceration, which starts from the mucous glands; it may occasionally give rise to warty growths, and always produces some thickening of the submucous tissue, with relaxation of the ligaments and muscles.

*Croupous Inflammation of the Larynx.**—The larynx is the most frequent seat of croupous exudation, and though occasionally found to extend even into the smaller bronchial ramifications, the deposit in the trachea and bronchi appears invariably to be continuous with the laryngeal exudation. In this form of inflammation false membranes are produced on the mucous surface, which cover it in more or less perfectly continuous layers. They are composed essentially of a delicate network of fine threads, entangling numerous mucous corpuscles or leucocytes. The threads have been regarded as fibrinous, and produced by the exudation of a coagulable substance from the blood-vessels; but it is probable that the true origin and nature of the false membrane are very different. It is usually of a light yellow or cream colour, varies from a mere film to a line and a half in thickness, and is more or less closely attached to the mucous membrane, from which in early stages it cannot be completely separated without wounding the surface. As the disease goes on, however, the layer of false membrane becomes loosened, leaving a brightly injected surface below which may, in some cases, show no further change.

Croup of the larynx and trachea is said by Rindfleisch to be distinguished by the stratified character of the false membrane; layers of roundish lymphoid cells alternating with homogeneous layers of what looks like fibrin; and he regards it as uncertain whether it is always (in this part) composed, as Wagner supposes, of altered cells. The secretion resembles that of a serous membrane in tenacity, adhesiveness, and coagulability. It is deposited in patches, which may gradually coalesce, and thus form rings or sheets of greater or less extent; they send off small prolongations into the follicles scattered over the mucous membrane, by which they are secured in their place until a suppurative process, underneath, loosens them, and allows them to be thrown off, if the patient retains strength to do so. There rarely are any abrasions of the mucous membrane of the trachea. If there is more than usual difficulty in removing the false membrane from the larynx, this depends, as Dr. West remarks, upon the more extensive alterations which this part of the air tube has undergone. It is generally red and swollen, especially about the edges of the rima glottidis, the arytænoid cartilages, and the openings of the sacculus laryngis. It is in the former that we occasionally find small aphthous ulcerations.

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* The reader is particularly referred to the 19th and 20th Lectures in Dr. West's work on the "Diseases of Infancy," &c.

Croup is a disease known by a great variety of names, which are calculated to embarrass the student. Cynanche is the oldest term, under which it is described by Paulus Ægineta as a well-known affection (see the Sydenham Society's Edition of his work, vol. i. p. 464); modern writers term it promiscuously cynanche, angina, trachitis, (with the epithets, membranacea, polyposa, stridula) asthma acutum infantum, &c. The English name croup is of Scotch origin, and was first, we believe, introduced into general use by Sir Everard Home. It has special reference to the sound produced in laryngeal obstruction. Croup and diphtheria have often been taken as identical, and the distinction between them is still involved in some obscurity.

The diseases with which croupy inflammation is most frequently complicated are bronchitis and pneumonia; though we very commonly meet with no other symptoms of morbid action than those found in the part affected; nor is there any doubt that these may be so slight as altogether to escape observation, in the post-mortem examination. We must here, as elsewhere, be careful not to mistake the pulmonic congestion, resulting from the suffocative influence of the malady, with inflammatory action.

Diphtheritic Inflammation of the Larynx.—This affection is not very common, and is usually caused by the extension of the diphtheritic process from the pharynx, fauces, or mouth. Still, in some epidemics of diphtheria, it is not rare, as was first clearly described by Bretonneau of Tours.* According to his observations, the exudation occurred simultaneously in the pharynx and air passages in fifty cases, while in one only the former was unaffected. Six or seven times he found that the false membrane extended to the smallest bronchi, and in one-third of the entire number it reached beyond the main division of the bronchi; in all the rest it terminated at different points of the trachea.

The distinction from croupous inflammation is usually clear anatomically, though in particular cases it may be impossible to distinguish them clinically. The diphtheritic false membrane cannot be separated from the mucous surface without removing more or less of the epithelium, or even some of the deeper structures; being in fact, partly composed of altered and necrotic tissue. The destructive process may extend to the submucous tissue or even to the muscles.

Ulceration of the Larynx.—The following kinds of ulcers are found:—

(1) *Catarrhal ulcers.* These are either produced by necrosis of the

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* "Des Inflammations Spéciales du Tissu muqueux," &c., par P. Bretonneau, p. 32. Paris, 1827.

mucous tissue itself, in which case they are small, flat, shallow depressions; or else arise in the mucous follicles, when they are deeper and more conical. Neither of these forms extends deeply or causes any serious destruction.

(2) *Syphilitic ulcers* are much more serious than those just mentioned. They begin mostly at the side of the epiglottis, and form sometimes very deep excavations. They have brightly injected and swollen edges from which warty growths often arise. If they heal they produce very hard contracting scars which may cause considerable narrowing of the passage. Sometimes the ulceration spreads very widely, over a large portion of the mucous surface of the larynx, and may extend to the trachea. Under the ulcers there is always much thickening and infiltration with lymphoid corpuscles.

(3) *Typhoid ulcers.* In the course of typhoid fever, ulcers sometimes form on the mucous surface of the larynx. They are (according to Förster) irregular, angular, deep, with villous margin and base; and arise from a new formation of small cells in the mucous membrane leading to sloughing. They are not usually deep, but still a remarkable case is recorded by Dr. Wilks,* in which an ulcer of this kind situated behind, at the junction of the vocal chords had quite perforated the wall, admitting air and mucus into a space in front of the œsophagus. The air which thus made its way into the posterior mediastinum penetrated into the thoracic walls, and produced general subcutaneous emphysema.

(4) *Variolous ulcers* are produced in the course of small-pox, by the formation and rupture of pustules on the mucous surface.

Ulceration of the larynx also occurs in glanders.

Tubercular or Phthisical Affections of the Larynx; Laryngeal Phthisis.—It is very common in cases of pulmonary phthisis to find catarrh, swelling of the follicular structures, and ulceration of the larynx. The latter has been generally described as tubercular, but much doubt exists whether the disease originates in or is even in most cases accompanied by the formation of tubercles, even when these are undoubtedly present in the lungs. Sometimes, though not commonly, tubercles are found in the parts surrounding the ulcer. Hence the greatest diversity of opinion has prevailed among pathologists.

These ulcers may be scarcely wider than a large pin's head, but by their confluence produce broad irregular patches. Their edges are often thickened or indurated, sometimes giving origin to papillary growths. The surrounding parts are injected, but less so than in syphilitic disease. Rokitansky states that tubercle is deposited in the form of grey granulations in the submucous areolar tissue, or as yellow caseous matter, infiltrated into the mucous membrane. He has found it constantly and exclusively in the vicinity of the transverse muscles and the adjacent arytænoid cartilages. His experience is strikingly at variance with that of Louis, who has not in a single instance met with tuberculous granulations in the substance, or on the surface of the epiglottis, larynx, or trachea. Dr. Watson attributes the ulcers of the larynx occurring in phthisis exclusively to the acridity of the sputa, but although the sputa manifestly exercise an influence in their production, there must be some further cause, as they are by no means constant accompaniments of phthisis; and as they do not form at some points, *e.g.*, the ventricles of the larynx, in which a lodgment of sputa must constantly take place. Louis states ulcers in the larynx to have occurred in a fourth part of his cases.

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* "Pathological Transactions," 1858, vol. ix. p. 34.

Förster, who gave much attention to this subject, thought that the doubts which had been expressed as to the nature of true tubercular disease in the larynx, had arisen from want of minute microscopical investigation. He regarded the ulcers as arising either from the necrosis of groups of miliary tubercles, or else from a diffuse infiltration. Virchow states that when a tubercle is situated superficially in a mucous membrane, its decay begins from the outside till it falls out leaving an ulcer. The absence of yellow granular or cheesy matter, which has led some to deny the analogy of this morbid process with tubercular phthisis, is to be explained by the rapid and early removal of the degenerated products. As to the situation of the ulcers, Förster* found among fifty carefully investigated cases, sixteen in which they were confined to the posterior commissure, and eight more in which the posterior commissure and posterior wall were the only parts affected, while in most of the others the affected area was more extensive. This shows a marked predilection of the posterior half of the larynx to this disease. In fifteen out of fifty cases, the trachea was likewise affected. This affection is almost always secondary to pulmonary phthisis; as a primary disease, it is exceedingly rare. This lends some support to a modification of the older view, that these ulcerations were produced by the sputa from a phthisical lung; since such products are admitted to be capable of producing tubercles by local infection.

In children, ulceration of the air passages is scarcely ever met with; Dr. West† has only seen it once in early life; on that occasion, several small excavated erosions were found, just above the chordæ vocales, in a child of twenty months, besides a general redness of the bronchial tubes. In the larynx their site is generally at the junction of the vocal chords, on the vocal chords themselves, and on the arytænoid cartilages.

Affections of deeper parts of the Larynx.—The submucous tissue, the cartilages and their perichondrium, are often affected by extension of disease from the mucous membrane, especially in cases of syphilitic or phthisical disease; and in this way extensive destruction of the deeper structures produced.

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* "Handbuch der Path. Anat.," vol. ii. p. 320. 2nd edition, 1863.

† Tubercular ulceration of the larynx in a case of general tuberculosis occurring in a child aged seven is reported in the "Pathological Transactions" for 1869, vol. xx. p. 33.

Primary perichondritis, or inflammation of the membrane covering the cartilages, occurs most often as a consequence of syphilis. It may give rise to an abscess, which causes even fatal obstruction of the glottis before it bursts; or to deep-seated ulceration and necrosis of the cartilage.* Abscess very rarely occurs independent of syphilis.†

The necrosed cartilage may be completely separated by suppuration and even expectorated during life.

New Growths in the Larynx.—These have lately received much attention since their examination has been facilitated by the laryngoscope. In the great majority of cases they are seated on the vocal chords. According to the statistics of Dr. Morell Mackenzie,‡ the most common form of growth is a papilloma or papillary outgrowth, consisting of an hypertrophied vascular papilla with more or less connective tissue, and covered with epithelium, sometimes sessile, sometimes pedunculated. These amounted to 67 per cent. in his list of cases (from which all cancers or malignant tumours were carefully excluded). Fibromata, including fibrous and so-called fibro-cellular tumours, come next, giving 16 per cent. These are generally pedunculated and make up the chief part of what have been called fibrous or mucous polypi. Other simple tumours, such as myxoma, lipoma, adenoma, angioma, and cystic growths have been very rarely observed, mostly in one or two instances only. Sarcoma is somewhat more frequent; nine cases of the spindle-celled variety having been observed or collected by Dr. Mackenzie. Simple epithelial growths, produced by mere hyperplasia of the epithelium, without any outgrowth of papillæ (as in papilloma), and sessile, constituted 5 per cent. of the cases.

Epithelioma is not common, though we cannot form any opinion as to its relative frequency as compared with other growths. It may form prominent outgrowths, but its more constant and more dangerous habit is to cause deep ulceration, which, after destroying the larynx, may perforate the œsophagus.

Cancer (scirrhous or medullary) is extremely rare.§

The laryngeal cartilages have a tendency to ossify in advanced life in obedience to the general law affecting similar structures; but this metamorphosis is also produced by disease; and it is probably in those cases of chronic laryngitis chiefly, which are connected with a rheumatic diathesis, and originate in the perichondrium, that this metamorphosis takes place. The larynx is not so liable to be affected by tumours or foreign bodies pressing upon it as other parts of the respiratory system, on account of the greater rigidity of its walls, and the capaciousness of its interior. Foreign bodies arrested in the œsophagus generally become impacted behind the larynx; when introduced by the glottis, they may become fixed in the chink; but if they pass the portal they necessarily drop through the wider cavity of the larynx, and become fixed at some lower point, or play up and down in the trachea.

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* "Trans. Path. Soc.," 1864, vol. xv. p. 36.

† "Trans. Path. Soc.," 1868, vol. xix. p. 74.

‡ "Essay on Growths in the Larynx." London, 1871.

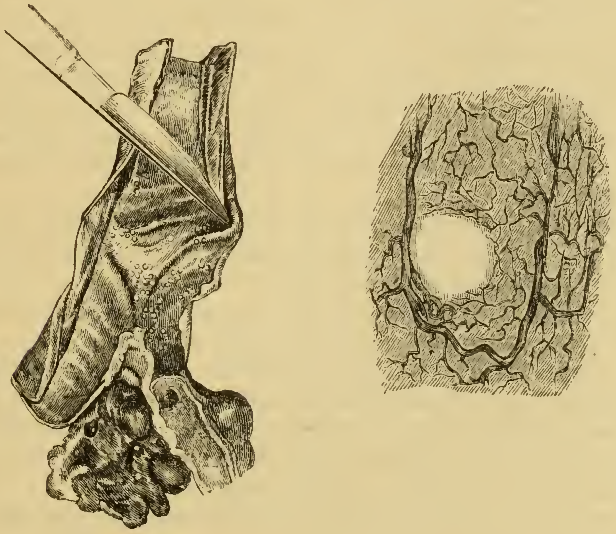
§ Cases of epithelioma of the larynx are recorded in the "Pathological Transactions," vol. x. p. 63; vol. xii, pp. 44, 104; vol. xiii. pp. 23, 28; vol. xvi. pp. 53, 54; vol. xvii. pp. 22, 33; vol. xviii. p. 25, &c. Primary cancer of the medullary kind is recorded by Dr. Broadbent, vol. xii. p. 44 (second case).

A singular accident which has been known to happen is eversion of the sacculus laryngis, producing a body precisely like a tumour.*

THE TRACHEA.

Many of the observations which we have had occasion to make on the pathology of the larynx apply to the trachea. The congestion of the mucous membrane presents similar characters; it is generally observed to be most marked in the membranous interstices, between the rings and at the posterior part. Occasionally it is bounded by a sharp outline; thus, where the hyperæmia of the trachea is associated with disease of one lung, it may be seen on

FIG. 98.



Enlargement of follicles of mucous membrane of the trachea, closely resembling miliary granulations, and accompanying pulmonary phthisis. The drawing on the right is an enlarged view of a single follicle, surrounded by enlarged and congested vessels. The glandular structure was distinctly apparent when examined under a higher power.

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* Moxon: "Path. Trans.," vol. xix. p. 65. Morell Mackenzie, *op. cit.* p. 34.

the corresponding half of the former only, the mesian line forming the division between the normal pale tissue and the portion that presents increased vascularity. A similar observation has been made with regard to the occurrence of ulcers, viz., that when following unilateral pulmonary disease, they are confined to the corresponding side of the trachea. Otherwise the point of divergence of the bronchi and the posterior mesian line are the most frequent seat of tracheal ulcers. They may often be traced to follicular inflammation, in the same way as obstructed solitary follicles put on the appearance of tubercular deposit. Enlargement of the follicles is far more characteristic of tracheal inflammation than of that of the larynx or bronchi.

Tubercular ulceration of the trachea rarely if ever occurs without the same disease of the larynx. The essential connection between ulceration of the trachea and pulmonary phthisis, is even more firmly established than in regard to the relation between the latter and laryngeal ulceration. Louis* found that of 190 phthisical subjects, seventy-six presented ulcerations in the trachea, while, in 500 non-phthisical subjects carried off by chronic diseases, examined by the same pathologist, not one presented any tracheal or laryngeal ulceration. A curious circumstance connected with this question is, the different ratio in which the two sexes are effected; thus, Louis has shown, that while only about one-fourth of the females who succumb to pulmonary consumption exhibit ulcers in the trachea, they are found in half the male subjects similarly diseased.

Diphtheritic and croupous inflammation of the trachea present no characters which differ from the phenomena exhibited by these diseases when affecting the larynx alone.

Syphilitic affections of the trachea are not common, except as an extension of the same disease affecting the larynx.

Ulceration of the trachea and bronchi, without participation of the larynx, has been sometimes, though very rarely, observed.

Perforation of the trachea sometimes occurs in consequence of extension of cancerous disease from the œsophagus, or simply from the pressure of an aneurism or other tumour.

New growths in general, polypi, epithelioma, and others, are far less common than in the larynx.

The rings of the trachea, like the cartilaginous structures of the larynx, are liable to become abnormally ossified, a condition which does not in itself exert any material influence on the health of the individual. The functions of the trachea are more liable to be interfered with than those of the upper portion of the air tubes, by pressure exerted from without; encysted tumours, hypertrophy, and other diseased conditions of the thyroid gland, aneurismal tumours of the carotid or subclavian arteries, or of the arch of the aorta, enlarged cervical glands, tumours, and foreign bodies in the œsophagus, phlegmonous erysipelas and diffuse abscesses in the vicinity, may each induce compression of the windpipe to such an extent as to cause death, or interfere with its functions by pressing on the recurrent laryngeal nerve. The practical question which arises in these cases regards the propriety of performing laryngotomy or tracheotomy; it is also one that requires great judgment in connection with disease of the mucous membrane, and the presence of foreign bodies within the tube.

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* "On Phthisis," Syd. Soc. Ed. p. 42.

Diverticula are described as occasionally forming in the trachea, by a dilatation of a portion of the posterior wall and consequent protrusion into the œsophagus between the ends of the tracheal rings; they have been attributed by Rokitansky to an hypertrophy of the muciparous glands. The dilated duct of the hypertrophied cysts may be discovered in the pouch; which confirms the explanation of its origin. When met with in the trachea, a similar condition is sometimes traceable in the bronchial tubes.

Foreign Bodies in the Trachea.—The presence of extraneous matters in the air passages is always an occurrence of very serious import; but unless the substance becomes impacted in the rima glottidis, it is not immediately fatal; the mucous membrane of the lower portions being less irritable and less liable to produce spasmodic action. The more rough the surface of the foreign body, the sooner inflammation is likely to supervene; and unless it be removed by coughing, or by an operative proceeding, the issue is certain death. If of a globular form, and too large to enter into the bronchi, it will pass up and down in the trachea, giving rise to a variety of sounds. Cherry-stones, buttons, coins, teeth, morsels of food, portions of bone and other things, have thus found their way into the trachea, and remained there or got impacted in the bronchi. Such bodies find their way, in an immense majority of cases, into the right bronchus rather than the left, owing to the former being a more direct continuation of the trachea than the latter. One case is recorded by Mr. Liston,* as a solitary instance, of a piece of bone having lodged in the right bronchus, and having been discovered and removed during life. A triangular piece of mutton-bone had, six months previous to the operation, become entangled in the glottis, and shortly after slipped down the trachea. A perfect recovery resulted.

Portions of food may repeatedly pass into the air passages in the case of persons whose power of swallowing is impaired, either from central paralysis, or from the pressure of tumours, aneurism, &c., on the nerves. The food will always pass by preference into the right bronchus, and set up pneumonia of the right lower lobe. The contents of the stomach may sometimes after death be forced up the œsophagus by distension of the intestines, and reach the same destination. Softening of the lung results if the stomach contained fresh gastric fluid, and may be mistaken for gangrene, but will be distinguished by the absence of the true gangrenous odour.

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* "Practical Surgery," 3rd ed. p. 412.

The upper portion of the air tube is also liable to fatal injuries inflicted accidentally or intentionally. In those cases in which life is not immediately destroyed by hæmorrhage, the injury may prove fatal by the secondary inflammation and tumefaction of the mucous membrane, or by coagula obstructing the passage of air. The latter is particularly likely to happen as a consequence of hasty surgical interference, in prematurely sewing up the wound, and thus preventing the necessary escape of the blood.

CHAPTER XXX.

THE BRONCHIAL TUBES.

THE diseases of the bronchial tubes affect the individual very differently, according as the larger or smaller divisions are the seat of the morbid action. Thus the same amount of tumefaction which, in the mucous membrane of the bronchi near the trachea, will scarcely give rise to any inconvenience, will, in the finer ramifications, be the cause of intense dyspnœa and danger. The terminal points of the respiratory system, in this respect, resemble each other; an acute tumefaction about the aperture of the larynx, and at the opposite end of the bronchial tree, may equally induce suffocative symptoms. Moreover, it is not always easy to determine the limits of bronchial and parenchymatous disease of the lungs; and it is scarcely possible that inflammatory affections of the latter can take place without involving the smaller bronchi.

Bronchial Hyperæmia and Hæmorrhage.—Hyperæmia of the bronchial mucous membrane is a phenomenon of everyday occurrence, accompanying catarrh, and gastro-intestinal, hepatic or cardiac affections, and manifested in its most marked forms by hæmorrhage. Hæmoptysis, which in the early or preliminary stages of phthisis is usually dependent on bronchial hæmorrhage, is rarely idiopathic as a mere result of plethora, but commonly associated with a deeper seated morbid affection; a crasis subsequently manifesting itself by further disorganization, or an organic disease, which has already been discovered. Pathologists have not yet succeeded in demonstrating the exact manner in which capillary hæmorrhage takes place; in a few instances of advanced tuberculosis, patulous vessels which were eroded by the progress of the ulceration, have been discovered, to which pulmonary hæmorrhage could be attributed; but the common process by which the blood is discharged, is perhaps analogous to the "sweating" of blood, by which the menstrual flow has been observed to be effected; it is rather by what has been termed exhalation or diapedesis that the overloaded vessels relieve themselves, than, as the term hæmorrhage* implies, by laceration or rupture. The researches of

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* Hæmorrhage, etymol. αἷμα, blood; ῥήγνυμι, I rupture; ῥαγὰς, a cleft, a fissure.

Cohnheim have shown the possibility of this method of extravasation. Hæmorrhage is not a mere passive occurrence, resulting from a retardation of the vascular current, or we should find it accompanying hypostatic congestion, which it does not; it must be looked upon as an evidence of further disease of an active character, as a symptom calling for our careful attention, but only to be treated in reference to a fundamental affection. Hæmorrhage from the lungs rarely proves fatal in itself, though at times the amount of blood lost is very considerable. Dr. Copland quotes the case of a patient who lost about 192 ounces in twenty-four hours and recovered. The frequency with which it is associated with phthisis, has been determined by Louis to be about two-thirds of all the cases; a relation which is confirmed by recent observers, and one which, taken with other circumstances, casts much suspicion on the nature of the cases of so-called vicarious hæmorrhages, whether they are supposed to take the place of the menstrual or hæmorrhoidal discharge.

The difficulty of explaining the mode in which hæmoptysis takes place, is as great in regard to the stages of softening as in the earlier stage of crudity; the vessels become obliterated in the former instance as the tubercular matter and pulmonary tissues deliquesce, and we rarely are able to discover the open mouths of lacerated vessels. At a future page we shall also have occasion to see that the hæmorrhage into the tissue of the organs constituting pulmonary apoplexy, is distinct from that bronchial hæmorrhage with which hæmoptysis is commonly associated. In either stage, therefore, of tubercular disease, it is possible that the discharge takes place from the bronchial mucous membrane. The relation of hæmoptysis to the different stages of phthisis, is one to which Dr. Walshe* has especially directed his attention; the results of an analysis of the cases which had occurred at the Hospital for Consumption at Brompton, in reference to this question, are as follow:

	Number of Cases.	Frequency of Hæmoptysis.	
		Absolute.	Per Cent.
First stage	39	28	71·79
Second stage	20	18	90·00
Third stage	69	57	82·61

The proportion changes somewhat, when the analysis is made for each of the sexes; we then find that the increase of hæmoptysis during the second and third stages is considerably greater in men than in women, which may fairly be explained by the greater bodily labour, and increased tax upon the pulmonary circulation, in the former than in the latter; it rather tends to show that the exciting influence of the tubercular cachexia itself, in producing the hæmorrhage, is greater than that of its secondary results. The following is the table illustrating this point:—

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* "Medico-Chirurgical Review," vol. iii. p. 225.

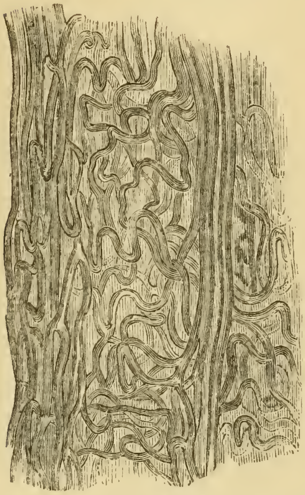
	Males.	Hæmoptysis.		Females.	Hæmoptysis.	
	Number of Cases.	Absolute frequency.	Per Cent. frequency.	Number of Cases.	Absolute frequency.	Per Cent. frequency.
1st stage	18	12	66·65	21	16	76·19
2nd and 3rd stages	56	49	87·50	33	26	78·78

Evidence of Hæmorrhage.—The anatomical signs after death of bleeding into the bronchi (from whatever source) are very distinct when once clearly appreciated. The blood in the trachea or larger bronchi is by the respiratory movements drawn into the smaller tubes, which it blocks up; the air which they contain, together with the residual air of the air-cells, being driven back into the latter and there imprisoned. When the hæmorrhage is recent, the lung tissue will accordingly be found over-distended, almost emphysematous and pale; the bronchi occupied by the blood forming bright red spots in it. Subsequently, however, the imprisoned air is absorbed and the tissue collapses, while the blood pigment becomes diffused into the parts surrounding the bronchial tubes, producing first a red, and ultimately a black staining. The irritation of the blood sometimes sets up inflammation in the form of bronchitis and catarrhal pneumonia.

BRONCHITIS.

In inflammation of the bronchi we find the same variations of injection and secretion that are presented to us in similar conditions of the upper portions of the air conduit; but the nearer we approach the terminations of the subdivisions, the more the bronchules will be found filled with the fluids poured into them, corresponding in character, in a measure, to the sputa seen before death, but with a greater admixture of air, the less viscid and tenacious the secretion. The post-mortem appearances may be limited to mere redness of the mucous membrane. In the smallest subdivisions we must be careful to discriminate between the injection of the bronchules and the redness resulting from the translucency of their tissue, allowing the colour of the subjacent pulmonary parenchyma to shine through. The redness is generally tolerably uniform in the part affected, fading off at the margin into the healthy tissue; we do not commonly meet with that arborescent or punctiform injection in the bronchi, which is seen in inflammations of other mucous membranes, as that of the stomach. Sometimes the affection resides exclusively in the larger bronchi, fading off in the smaller divisions; at others it occupies the reverse relation; the danger to the individual increasing with the number of small tubes affected; the tumefaction and loss of elasticity in which, necessarily exert a great influence in producing dyspnœa. The actual sense of the difficulty of breathing, as well as the real absence of proper aëration of the blood, shown by the lividity of the patient, has appeared to us to be greater in these cases of capillary bronchitis than in pneumonia. The more asthenic the form of bronchitis, the more the redness of the bronchial mucous membrane approaches a livid purplish tint; it is generally found of this hue in the chronic forms. The secretions will vary according to the stage and character of the disease, from a viscid glazy mucus, to a genuine purulent discharge of a more or less diffluent character. Occasionally, death is the result of a sudden effusion of liquid into the bronchi, constituting what is called suffocative catarrh, which is met with more frequently in the infant than in the adult. Long-continued purulent expectoration may, however, have existed during life, without any appreciable lesion being discovered after death; in these cases, the bronchial mucous membrane, as Andral* observes, need not even present a trace of redness. In examining the lungs we must be careful to compare different portions, before arriving at definite conclusions; for it is often difficult to determine to which part the fluids belong which exude on section. The surest way to ascertain the state of the parietes and contents of the bronchi, is to follow them from the larger trunks with a pair of scissors, being careful not to admit more extraneous matter into the tubes than we can help.

Fig. 99.



Injection and stasis in the vessels of the bronchial mucous membrane, in bronchitis, seen by a low power. The vessels were disposed in longitudinal clusters united by transverse inosculations.

Plastic Bronchitis; Bronchial Polypi.—True croupy inflammation may affect the bronchi, as it does the upper respiratory passages, though it is met with less frequently; complete moulds of portions of the bronchial tree present themselves to us; the influence they exert upon respiration depends partly upon the obstruction they themselves offer, and partly upon the tumefaction of the subjacent mucous membrane.

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* "Précis d'Anatomie Pathologique," vol. ii. p. 481.

The mucous membrane of the bronchi, like the mucous membrane of the urino-genital organs, occasionally exhibits a chronic affection of this kind, in which, without marked symptoms of an inflammatory character, the membrane pours out a plastic exudation, which forms, what have been termed, bronchial polypi; that is to say, more or less complete moulds of the bronchial tubes, sometimes solid or even laminated, sometimes hollow. These casts are usually expectorated in a curiously convoluted form, being rolled up, as it were, into pellets during the act of expectoration; but when placed in water, expand into an arborescent form, corresponding to the ramifications of the bronchi. The process sometimes extends into the very smallest tubes, and we have seen the casts terminated by a rough mould of the alveolar spaces, a fact which connects this affection with pneumonia. The "polypi" are composed of a firm network of fibrin, enclosing numerous leucocytes and some few red corpuscles; so that in structure they resemble the pneumonic exudation. Their expulsion is often accompanied or preceded by bronchial hæmorrhage, but there is not sufficient reason for concluding, as some have done, that the casts themselves are composed of coagulated blood. Sir Thomas Watson observes, that though it is surprising that patients should recover from the affection, it never in itself seems to prove fatal. Dr. Reid* has reported two cases of tubular expectoration from the bronchi occurring in the adult, with delineations, which closely resemble that given by Dr. Baillie, in his work on Morbid Anatomy. In one, the patient, a married lady, aged twenty-eight, affected with a chronic cough, consequent upon an attack of bronchitis, frequently, after suffocative attacks, coughed up arborescent membranous substances, resembling casts of the minute bronchial tubes; the second case occurred in a gentleman, aged forty-four, and closely resembled the former, except that there was more manifest congestion, and that the casts were more of a sanguineous character, and their rejection each time accompanied by some hæmorrhage. It appears to be more frequent on the continent of Europe, where the affection has carried off several distinguished individuals, among whom the Empress Josephine is the best known.

Dr. Peacock some years ago analyzed more than thirty recorded cases of this disease, and found that the great majority occurred in the male sex, and in persons in middle life. In about half the cases there was hæmorrhage, which in ten was very profuse. In many cases some other disease, as phthisis or pneumonia, was present, and where death occurred it was usually due to this cause, not to the bronchial affection.†

There are some difficulties in the way of conceiving a fibrinous exudation being produced upon such a surface as the bronchial mucous membrane when it is found (as it usually is) quite unaltered. It has, therefore, been urged by Dr. Bristowe* that in some cases the exudation is not produced by the mucous membrane, but coagulable material effused into the air-cells finds its way into the smaller bronchial tubes, and is there moulded into the shape in which it is found. The material is certainly identical with that which fills the air-vesicles in pneumonia. We may repeat that we have seen small casts terminated by a rough mould of the alveolar spaces, such as is seen in pneumonia. Moreover, in the latter complaint it is not unusual to find small masses of lymph prolonged into the bronchial tubes for the distance of half or a quarter of an inch. This explanation is therefore extremely plausible in those cases in which the fibrinous bronchial casts accompany pneumonia. The casts are chiefly found in the smaller bronchi, up to those as large as a quill; and then extend continuously into the smaller ramifications. Numerous cases of this affection are recorded in the Transactions of the Pathological Society.† They cannot always be clearly separated from laryngeal or tracheal croup extending to the bronchi.

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* "Medico-Chirurgical Transactions," vol. xxxvii. p. 333.

† "Trans. Path. Soc." vol. v. p. 41.

Diphtheria sometimes attacks the bronchial mucous membrane; but so far as is yet known, only subsequently to the same disease in the larynx and trachea. A case of this kind is recorded by Dr. Murchison‡ in which the larynx, trachea, and bronchi, down to their fine ramifications, were lined with a thick false membrane, forming a perfect cast of these tubes, but easily detached from the subjacent membrane, which was intensely injected. The false membrane was everywhere made up of altered epithelial cells without any fibrillated structure.§ In a very similar case, described as croup, but in which there were false membranes on the tonsils, the false membranes extended in part of the lung to the finest bronchi, starting with the larynx and trachea.‖ We have lately seen a perfectly analogous case, in which the clinical symptoms were those of diphtheria.

ULCERATION OF THE BRONCHI.

Simple inflammation of the bronchi without any specific character may undoubtedly lead to ulceration. The "catarrhal" ulcers thus formed are small, shallow, and confined to the epithelial structures. Deep ulceration extending to the submucous tissue, or still deeper, also occurs; but this, as in the larynx and trachea, is mainly a concomitant of phthisis. It has been observed by few authors, probably on account of the care requisite in the examination, to discover an abrasion in these parts. Louis, who examined the bronchi of forty-nine phthisical subjects, with special regard to this point, found ulcerations in twenty-two. Dr. Copland is of opinion that ulcers occasionally perforate the bronchial tubes, and thus occasion abscesses in the pulmonary parenchyma, and there is no doubt that this is one of the processes by which cavities in phthisis are produced. In these cases there appears to be an antecedent production of tubercles in the mucous membrane, and tubercles may often be seen in the neighbourhood of such an ulcer. If they are wanting, it is still possible that the ulcer may have been originally tubercular; but no perfectly definite rule can be laid down to distinguish tubercular from simple catarrhal ulcers.

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* "Trans. Path. Soc." vol. vi. p. 59.

† "Trans. Path. Soc." vol. v. p. 41; (statistics) vol. vi. pp. 59, 68; vol. vii. pp. 54, 56; vol. xvi. p. 48; vol. xvii. p. 29; vol. xxi. p. 64.

‡ "Pathological Transactions," 1871, vol. xxii. p. 35.

§ This would doubtless, by some pathologists, be described as a case of croup.

‖ "Trans. Path. Soc." vol. x. p. 69.

It has also been supposed that there is a syphilitic ulceration of the bronchi, as of the trachea and larynx, but this hardly seems to be established with certainty. Some instances of extensive ulceration, independent of tubercle or any specific disease, are, however, recorded. In a case of this kind observed by the editor* the surface of nearly all the bronchi of both lungs as well as the trachea, but not the larynx, was covered with ulcers, sharply defined and of irregular shape. They extended nearly through the mucous membrane, and appeared to have their starting-point in an infiltration with lymphoid cells which affected all the mucous and submucous tissue, both of the cartilaginous and non-cartilaginous parts of the wall; and was unconnected either with the mucous glands, the cartilage, or the perichondrium. It had much resemblance to syphilitic ulceration, though it could not be definitely shown to have that character.

Obstruction of the Bronchi.—Other morbid states, besides those already alluded to, may give rise to obstruction or stenosis of the bronchial tubes; it may be produced by an actual hypertrophy of the submucous layers, as a result of chronic bronchial irritation, or by serous effusion, as in dropsical states of the system. The physical symptoms in these different cases may be identical, being produced by analogous structural alterations; but it is manifest that the constitutional basis upon which they rest may differ very considerably, as also the influence they exert in living subjects upon the further production of morbid conditions.

Typhoid Affections.—The pulmonic symptoms accompanying typhoid fever are referred by Rokitansky† to catarrh of the mucous membrane; he states it always to appear as an intense diffused congestion; the mucous membrane is of a dark, almost violet tint, swollen and succulent, and yields a secretion of a gelatinous and sometimes dark, blood-streaked mucus. The disease, according to this author, is most commonly developed in the bronchial ramifications of the lower lobes; it is always limited to the stage of congestion, and never gives rise to any apparent production of a secondary formation in the tissue of the mucous membrane, such as is produced in immense quantity in the intestinal follicles in cases of "abdominal typhus" (*i.e.*, typhoid fever).

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* "Pathological Transactions," 1869, vol. xx, p. 30, and pl. 1.

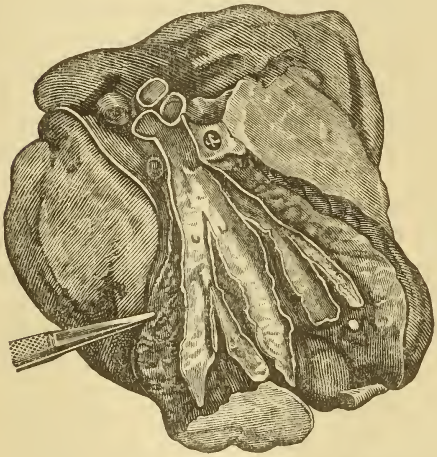
† "Pathological Anatomy," vol. iv. p. 23, Syd. Soc. Ed.

The submucous tissues may be variously affected in the bronchi, as in air-passages already considered. The cartilaginous rings may undergo a process of softening or become ossified; in the latter case, they become brittle, and break; they then either project like fish-bones, as Andral observes, into the bronchial cavity, or, becoming detached, are expectorated.

DILATATION OF THE BRONCHI.

Many of the lesions to which we have adverted may co-operate in producing a morbid condition of the bronchi, to which Laennec was the first to draw attention, and which, though of extreme importance in a nosological point of view, has hitherto been treated rather as a question affecting the pathologist than the practitioner. It is dilatation of the bronchi or bronchiectasis.* By Laennec it was attributed exclusively to an accumulation of mucus in the ramifications of the dilated portion; but, as Dr. C. J. B. Williams justly observes, if this were the cause we should not, as we do, hear the air penetrate freely into the dilated portions. There may, undoubtedly, be various efficient causes at play in the production of this diseased state, both of a mechanical and of a more dynamical character. The most palpable instance of the former is presented to us in the case of an enlarged bronchial gland, whether it contains cretaceous or simply scrofulous matter, compressing a bronchus. Here, the free exit of the respired atmosphere being prevented, an accumulation of air might be supposed

FIG. 100.



Dilated bronchi, from a female aged fifty-two, who had suffered from chronic pneumonia and bronchitis for three years; the pulmonary tissue intervening between the bronchi was much condensed.

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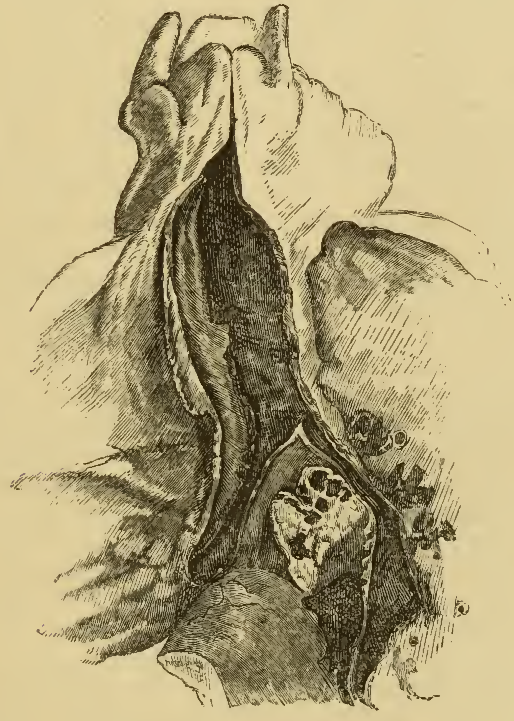
* Etymology,—*βρόγχια*, the bronchi, and ἔκτασις, dilatation, from ἐκτείνω, I stretch. We may take this opportunity of remarking upon the distinction which some authors have lately made between bronchia and bronchi; it is a source of some confusion, and scarcely warranted by their etymology of the words; if a diminution is required, the term bronchule is more convenient, and not liable to be mistaken.

to take place behind the narrowed portal, the channels being prevented ever collapsing to the same extent as a healthy lung. Any impediment to the entrance or exit of the air into the lungs will produce irregular and forcible breathing, and throw a greater strain upon those parts especially, which are in the vicinity of the obstacle.

One point having yielded, it is quite intelligible that the distension should gradually progress, while, at the same time, it must, in a corresponding ratio, compress and gradually obliterate the surrounding pulmonary tissue. In the majority of instances a diseased condition of the bronchial parietes, if not, as Corrigan has suggested, of the pulmonary parenchyma itself, precedes the occurrence of bronchiectasis.* When the changes have taken

Fig. 101.



Cretaceous enlargement of a bronchial gland, compressing the right bronchus, which is much dilated beyond the point.

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* Corrigan views the condition of the pulmonary tissues, giving rise to bronchial dilatations, as analogous to cirrhosis of the liver; he considers it the result of the formation of an unyielding fibrous tissue, to which the bronchi become attached, and therefore incapable of collapsing.

place in the tissues which are likely to give rise to it, any violent effort to distend the lungs, as in catarrh, bronchitis, or hooping-cough, may be the exciting cause.

Forms of Bronchial Dilatation.—The three forms which Laennec* describes have been successively adopted with some modifications by subsequent writers, though none have been satisfied with his rationale. In the first there is a solitary cystic dilatation or a series of distinct dilatations of a more or less spherical form, commonly affecting bronchi of the third and fourth order; these forms are essentially the same, and they generally present considerable attenuation of the dilated portions, while the intervening parts of the bronchi remain normal. The second (or Laennec's third form) differs entirely from those just considered, and consists in an almost uniform or cylindrical expansion of a single tube, or an entire section of the bronchial tree. The third form of some other writers, as Förster, is that of fusiform dilatation, constituting a transition between the two just mentioned. It is in the cylindrical dilatation that we meet with thickening of the parietes, tumefaction of the mucous membrane, which may be thrown into folds resembling those of the small intestine, and a proportionate increase in the subjacent fibrous tissue. When the dilatation affects the apex of the lung, it may proceed to such an extent as to resemble a tuberculous multilocular cavity. The perfect continuity of the mucous lining with that of the adjoining bronchi, and the smoothness of the tissues, will aid in determining the nature of the lesion, if there be any doubt.

The first form or saccular dilatation is not very common. The sacs may be as large as a walnut, or more rarely the size of an egg or larger. The bronchus opens into the cavity sometimes with its full width; sometimes by a constricted opening, which may be even altogether obliterated, so that the cavity represents a closed cyst. The cavity contains mucus and pus, sometimes extremely fetid. When there are several such cavities they may occupy a great part of the lung, and produce during life all the physical signs of phthisical vomicæ.†

The cylindrical form of dilatation is the most common, and is sometimes pretty general in both lungs. It is not seen in the larger bronchi, but begins mostly in those of the third order. These tubes, instead of becoming smaller, as they would normally do, retain a uniform diameter or become larger, and preserve this enlargement till close under the pleura, where they end mostly in a blunt rounded form.

A third variety, the fusiform, has been described, the tubes being unequally dilated in a spindle-shaped manner. It differs, however, but little from that last described, constituting perhaps a transition to the sacculated variety. It is very uncommon.

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* See Laennec's "Traité de l'Auscultation Médicale," &c., 1826, vol. i. p. 206.

† "Trans. Path. Soc.," vol. vi. p. 57.

It appears from Dr. West's description that in children the fusiform variety of bronchial dilatation is rarely, if ever found, while the cylindrical form is a common result of bronchitis. On one occasion, however, he saw a case in which, in addition to a general cylindrical enlargement of the tubes, many of them presented a marked dilatation, about half an inch from their termination, the tube expanding into a cavity big enough to hold half a large nut. The lining mucous membrane presented an extraordinary degree of thickening.

Condition of the Walls.—The walls of the dilated bronchial tubes vary much as to the degree and manner in which they are affected. Sometimes they are much wasted and attenuated, less commonly thickened—the latter chiefly in the saccular form. The elastic fibres are (according to Förster) always diminished, and the increased thickness, which is only found in chronic cases where the dilatation has existed a long time, is said to be always due to hypertrophy of fibrous tissue. The smooth muscle-fibres of the bronchial tubes are said by the same authority to be unaltered in the thickened walls. The inner surface is often uneven, being traversed by ridges, which leave deep recesses between them. The mucous membrane is usually in a state of chronic inflammation, being swollen, injected, and covered with purulent secretion. Occasionally the suppuration extends to the deeper tissue, destroying the wall and causing ulceration, from which ultimately a cavity may result.

Condition of the Pulmonary Tissue.—The surrounding parenchyma of the lung presents two orders of changes, the one thought to be connected with the production of bronchial dilatation, the other resulting from it. In the former case there is induration, imperfect expansion, or contraction of the lung tissue; and putting the case conversely, it may be said that whenever these conditions are present there is more or less dilatation of the bronchial tubes. This sequence is seen in phthisical conditions of the lung, in chronic pneumonia, and especially in that peculiar condition called by Corrigan cirrhosis of the lung. Wasting of the lung tissue is very common, and sometimes reaches an extreme degree, so that all the tissue intervening between the dilated tubes disappears, and the latter come in contact, or even form communications one with the other. This is usually set down as a consequence of the dilatation, but since atrophy often results from the conditions just mentioned, which lead to induration and contraction, it is not certain that so clear a line should be drawn between the two. When induration takes place, destruction of the lung, gangrene, and perforation of the pleura, with its consequences, may result.

Causes of Dilatation of the Bronchi.—By far the commonest antecedent condition is bronchitis, especially a chronic form of it, and this supplies at once two factors which are doubtless concerned in the production of dilatation, viz., relaxation of the walls, which commonly results from chronic inflammation, and increased pressure from habitual coughing. The other conditions already mentioned are usually present when the dilatation is extreme. Corrigan, who first drew attention to this point, suggested that the contracting pulmonary tissue would draw aside the walls of the bronchial tubes, and thus enlarge them. It is not quite easy to see how this should be, but it is certain that the contraction of the lung "tends to produce a vacuum," that is to say, diminishes the resistance to atmospheric pressure. This will then act externally, so as to produce falling in of the chest walls, and internally on the respiratory surface, so as to produce dilatation of all the air cavities. The conditions are thus the same as lead to emphysema, and will be further considered under that head. Dr. Wilkes states that the dilatation of the tubes occurs usually in children, that of the air vesicles or emphysema in adults.* Other conditions favouring dilatation in the same way are unequal distribution of pneumatic pressure from collapse, or airlessness of one portion of the lung, and impaired movement from pleuritic adhesions, or other impediments.

Tubercle and Scrofulous Products.—Tubercle is sometimes met with in the main bronchi, accompanying the same affection of the larynx or trachea in cases of general miliary tuberculosis. There are then miliary granulations upon the mucous surface. In the smaller tubes the growth appears to be situated rather in the walls. In chronic phthisis also, when tubercles are formed in the lung, they frequently occur in the walls of bronchial tubes, forming what has been called by Virchow peribronchitis tuberculosa, of which we shall speak again. Granulations may also sometimes be seen on the mucous surface, accompanied by ulcerations, and, in some cases, by yellow pasty matter. The latter is, however, believed by Virchow and others to be unconnected with tubercle, and to be the product of "scrofulous" bronchitis. We shall return to this subject in speaking of tubercle in the lung.

It seems clear that ulceration and destruction of the bronchial wall may result from the formation of tubercle, and that this may be one mode of origin of vomicæ.

Calcareous concretions derived from obsolete scrofulous or tubercular processes, are sometimes met with in the bronchi. What is called "Bronchial Phthisis," *i.e.*, caseous or tubercular softening of the bronchial glands in children, has been attributed to scrofulous or tubercular disease of the bronchi; but in most cases there is no proof of this.

Foreign Bodies.—Foreign bodies of the most varied kind may be found in the bronchial tubes. We need not speak of objects accidentally introduced into the air tubes which will, as we have said, by preference fall into the right bronchus. Food, whether swallowed or vomited, may sometimes pass into the bronchi, as may other contents of the stomach, often recognized by their smell. Blood, derived from hæmorrhage in any part of the respiratory tract, in the nose, mouth, or stomach, or from the bursting of an aneurism into any of these parts, may also be found; as well as pus from pulmonary abscesses or vomicæ, from empyema, or even from hepatic abscess (the latter stained with bile) and other products of inflammation. Parasites, if they are seen at all, are derived from elsewhere, since they are not in man, as in some of the lower animals, found in the bronchial tubes.

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* "Trans. Path. Soc.," 1862, vol. xiii. p. 28.

CHAPTER XXXI.

THE LUNGS.

Having in the foregoing pages considered the diseases of the respiratory passages, we now arrive at the investigation of the morbid changes which occur in the lungs themselves; in the tissues aiding in the purposes of oxygenation, the ultimate vesicular terminations of the bronchi and the interlobular tissue. The bronchules, long before their termination, are deprived of their cartilaginous rings; these are reduced to mere flakes before they cease altogether, and all trace of them disappears in tubes of less than one-sixth to one-tenth of an inch in diameter. The tracheal muscular fibres, on the other hand, are continued even to the terminal bronchules, but instead of merely filling up the gap in the cartilaginous frame-work, they form a uniform layer encircling the canal, but excessively thin. They may be seen in tubes not more than one-eightieth of an inch in diameter. Within the muscular layer are very thin longitudinal or elastic fibres; the ciliated epithelium descends into all but the terminal bronchules. The latter are, however, lined by a simple pavement epithelium, which most observers have traced into the air vesicles also. According to others, there is absolutely no epithelial covering in the air cells; and others, again, have been able to see nothing but nuclei. Without expressing any opinion as to the structure of a perfectly healthy lung, we must say that the existence of a continuous epithelium lining the air vesicles of lungs in the most varied pathological conditions appears to us incontestable. This epithelium rests upon an extremely delicate basement membrane, by which alone it is separated from the very rich and closely knit capillary network. The capillary vessels cover nearly the whole area of the alveolar surface, and when seen in profile, obviously project into the cavity; a fact of some importance. Another point in the anatomy of the lung, which must not be forgotten, is the relation of the bronchi to the air cells. The ultimate respiratory spaces or loculi are conical spaces, expanding from the smallest bronchial tubes, so that their opening is smaller than the cavity itself. On the walls of these cavities, which have been called infundibula (Rossignol) or intercellular passages (Rainey) are situated the air cells, which are hemispherical structures, and therefore open very widely into the common infundibular space, the septa dividing the alveoli being very shallow. In an earlier stage of development, as seen in the fœtal lung, the alveoli are grouped around the infundibulum like the acini of a racemose gland, so that the whole lung may, in fact, be compared to a complicated gland. This analogy, though it becomes fainter in the adult structure, is still of much importance, both as regards anatomy and physiology, and must not be forgotten in considering the morbid conditions of the air cells. Finally, we must state that the walls and septa of the air cells contain elastic fibres, and also (though in small quantity) ordinary connective tissue. It has also often been stated that the walls contain smooth muscular fibres, but this point is not clearly established. The anatomical structures just described, viz., the group of air vesicles and the infundibular passage, form a pathological as well as a physiological unity; it is, in fact, the morbid changes of these structures that we shall consider in the following chapters. In those changes the epithelium, the blood-vessels, and the connective tissues of the walls may severally be involved.

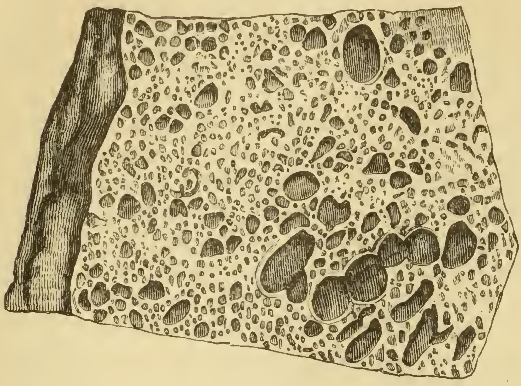
EMPHYSEMA.

The term emphysema is used in several senses. First as *interlobular emphysema.* This consists in effusion of air into the interstitial or interlobular tissues of the lung, from laceration or rupture of the air vesicles or bronchial tubes. This usually gives rise to a similar affection of the pleura, and may extend to the subcutaneous tissue of the neck, thorax, and body generally. It is well known that a similar condition of the subcutaneous tissue may arise from laceration of the trachea. These occurrences are comparatively rare, and not important. Development of gas in the body from putrefaction after death is sometimes called *post-mortem emphysema*, a condition only mentioned to prevent its being confounded with that of which we have now to speak.

VESICULAR EMPHYSEMA.

Dr. Baillie, though unable to suggest the means of distinguishing this disease before death, was one of the first to show its true nature in the dead body. It consists essentially in a dilatation of a larger or smaller number of air vesicles, and may be produced by any cause exerting a great strain upon them. The effect is to diminish the specific gravity of the part affected, so as to render it more buoyant than the healthy lung tissue in water; to cause the lung to become less crepitant on compression, giving it a doughy or woolly feel, to prevent its collapse on the thorax being opened, and to render it more or less dry and exsanguine. The emphysematous portion, if superficial, projects above the surface of the unaffected part; and large bullæ may be visible on the surface of the lung, from the gradual obliteration of the intervesicular tissue, allowing several vesicles to unite. The loss of elasticity in the pulmonary tissue, whether primary or secondary, prevents the usual collapse of the lung at the period of expiration; the vitiated air is not expelled as it ought to be; and this is one cause of want of oxygen. Another cause of deficient respiration is the diminution of the respiratory surface. As the groups of air cells expand and their partitions become obliterated, and still more when the walls are ruptured, two or more cavities being thrown into one, the area of the inner surface will become less.

Fig. 102.



Portion of emphysematous lung—the cavities are either formed by simply enlarged air cells, or by the coalescence of groups of cells.

(St. Bartholomew's Museum, xiv. 11.)

Minute Changes in Vesicular Emphysema.—The principal change seen in fine sections of the lung in an early stage of emphysema consists in enlargement of the central cavity or infundibulum of the terminal group of air cells, by which the air cells opening into it are removed further from one another, and from the centre. The peculiar appearance thus produced is seen very well in the representation of pneumonia given in Fig. 105. The lung from which the preparation was made, though hepatized, showed, even to the naked eye, enlargement of the air cells. Following this or at the same time, the partitions between the adjoining air cells of the same lobule become lower, the air cells shallower, and finally they all become merged in one cavity. This is evidently a return to the simpler type of lung found in the lower vertebrata, and as has been referred to above, the area of the interior surface becomes less. It will be easily seen how, if the excess of pressure and atrophy of the walls continue, communications will be formed between adjacent air cells (the existence of which normally is doubtful) and between adjacent lobules (which never exist normally). The larger cavities thus formed show in their walls deficiency of elastic fibres, but an excess of connective tissue; and as is stated by some observers, a superabundance of the connective tissue nuclei. The blood-vessels are also found to be scanty, being compressed and obliterated by pressure, and it is probably the blocking up of vessels containing blood, which produces the abundant pigmentation which the walls of the cavities also show.

Form of the Thorax in Emphysema.—The following is the description given by Sir W. Jenner:—The thorax is barrel-shaped; the antero-posterior lateral and vertical diameters are increased; the sternum is arched; the lower cervical, dorsal, and upper lumbar spine is curved, with the concavity forward; the ribs are too horizontal; the intercostal spaces are widened, and but little, if at all, depressed below the level of the ribs; the posterior bulgings on either side of the vertebral column are greater than they should be; the costal angle is larger than in health; the diaphragm flattened, and the lower part of the sternum forced forward.

Varieties of Emphysema.—Emphysema is in most cases confined to certain parts of the lung. The parts most commonly affected are the anterior borders of the upper lobes on both sides, and less generally other peripheral parts, as the lower borders of each lobe. This partial emphysema occurs in lungs, of which some part is impermeable to air through hepatization, compression, infiltration, induration, or any other cause. Such a condition is in all probability often produced during the last hours of life, or the death struggle by the violent efforts of inspiration; and is more often present than absent in post-mortem examinations. This variety has been called acute, vicarious or compensatory emphysema, but the same condition is produced chronically when parts of the lung are from any cause impermeable to air, constituting chronic vicarious emphysema. In both the acute and chronic conditions just mentioned it is hard to draw the line between true emphysema and simple distension of the lung.

Chronic Substantive Emphysema.—The condition known as actual or substantive emphysema is produced by chronic processes during life, and is much more important than that just described. This is but very rarely spread over the whole lung; more generally one lobe only is affected, and especially the peripheral parts of this. General emphysema of both lungs is probably quite unknown, or at least excessively rare. In all these cases the morbid condition predominates very much at the parts above mentioned. It is also often localized at certain spots where there is condensation and contraction of the lung, as is often the case at the apex.

Senile or Atrophic Emphysema.—Although atrophy of the walls of the air cells cannot be regarded as the primary change in ordinary emphysema, there is a form of the disease in which this appears to be the case. It occurs especially, or even exclusively in old age, and is accompanied by a diminution instead of increase in the size of the lung. Hence Sir W. Jenner distinguishes this form as "small-lunged emphysema." The lungs collapse very completely when the chest is opened, so much so that in Jenner's words "the weight of the lung may be sufficient to cause it to fall in like an inflated bag of wet paper." The air vesicles are in this form not distended, but by the absorption of the partitions several may be thrown into one. The whole solid tissue of the lung is remarkably diminished, and at the same time very deeply pigmented. This condition is described by many authors simply as atrophy of the lung.

Causes of Emphysema.—The credit of offering the first rational explanation of asthma, and its connection with emphysema, is due to Dr. Floyer,* though the priority of the discovery is commonly attributed to Laennec.

The explanations which have been given in later times may be considered under two heads.

In the first place it has been supposed that if the tissues are altered so as to lose their power of resistance, a normal pressure of air will cause excessive expansion. Mr. Rainey, in one of his interesting papers in the "Medico-Chirurgical Transactions,"† has attempted to show that fatty degeneration of the walls of the air cells exists in emphysema, and that the over-distension is due to the weakening of these structures thus produced. No *à priori* objection can be brought against the reasonableness of this explanation. It is, however, opposed by the fact that atrophy and fatty degeneration are not found in early but in later stages of emphysema, and are thus to be regarded rather as a consequence than as a cause of that condition. In the second place it has been held that while the tissues are unchanged excess of pressure on the inner surface of the air vesicles will cause first over-distension, and afterwards atrophy of their walls, with all the other changes met with in emphysematous lungs. This cause is obviously sufficient if it can be shown to exist, and much ingenuity and research have been spent in showing how such an excess of pressure comes to exist in the whole lung or in particular parts of it. Some have supposed the excessive strain to occur during inspiration only; others during expiration. There can be little doubt that it may be produced by both movements.

The first inspiratory theory was that of Laennec. He attributed the chief influence to hindrances to the exit of air from the lungs, such as the pressure of mucus in the smaller bronchi, swelling of their mucous membrane, pressure of tumours or enlarged glands on the larger bronchi, &c. In all such cases he thought air would be freely drawn in during inspiration, but being unable to escape would accumulate behind the seat of obstruction, while every successive inspiratory movement would increase the tension. Such a valve-like action is difficult of comprehension, because in general a plug that prevents the exit of air is even more likely to prevent its admission. It receives some support from the fact that when blood or other fluid is recently effused into the bronchi something like an emphysematous condition is found, probably because the inspiratory movement sucks the foreign substance further and further in, so as to imprison the residual air. But as Dr. Gairdner has pointed out, the ultimate result of obstruction of bronchi is collapse of the corresponding portion of lung.

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* In his little work on "Asthma," published in 1698, Dr. Floyer says: "The broken wind results from the rupture or dilatation of the bladders of the lungs, by which the air is too much retained in the bladders or their interstices, and thereby produce a permanent flatulent tumour in the whole substance of the lungs. It is not easy to explain the production of a permanent flatulent tumour in the lungs by a strain in running, but by supposing the bladders of the trachea too much distended, and the muscular fibres which constringe them in expiration, thereby over-stretched and made unfit to express the air afterwards; so that these bladders retaining more air than is usual, the substance of the lungs must appear always inflated."

† "Med.-Chir. Trans." vol. xxiii. p. 37.

Another inspiratory theory is that of Dr. Gairdner.* He has shown that emphysema of one part of the lung is always accompanied by collapse, atrophy, or consolidation of some other part. In a word, one part occupies more than its normal volume while another occupies less. Thus while the chest expands to its normal extent during inspiration, the pressure of the air drawn in will distend those parts into which it enters, so as to compensate for the non-expansion of those parts into which it does not enter. In other words, a normal bulk of air being drawn in, if certain parts of the lung are impermeable, the pressure on the remaining permeable parts will be excessive.

This explanation is unimpeachable as far as it goes, but it fails to explain the special distribution of emphysema in the lung, and its preponderance along the anterior edges and at the apex. These facts are accounted for on the expiratory theory, first propounded in this country by Sir W. Jenner (in 1857)† and in Germany by Ziemmsen and Mendelsohn (in 1845).‡

The chief merit of this theory consists in showing how the uniform pressure of expiration may give rise to unequal tension in different parts of the lung. If any obstacle exists to the passage of air outwards, as in expiratory efforts with closed glottis (coughing or muscular exertion), the internal pressure in all parts will be increased, but it will only produce excessive expansion where the external resistance of the thoracic walls is least. This will be the case at the apex, where the lung runs out of the thorax, and along the anterior edge, where the elasticity of the costal cartilages and the mobility of the sternum diminish the power of resistance. Hence these parts will, in excessive respiratory tension, suffer undue expansion. If any part of the lung is impermeable to air, the pressure of expiration will, like that of inspiration, cause excessive tension in those parts to which air has access. Adhesions of the pleura, preventing free movement of the lung, will also give rise indirectly to inequality of pressure. Over-distension once produced will not be permanent, unless some changes occur in the distended lung tissue. The first alteration is doubtless loss of elasticity, owing to which the dilated air spaces are not emptied.

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* "Brit. and Foreign Medico-Chirurgical Review," April, 1853, p. 452.

† "Medico-Chirurg. Trans.," 1857; Reynolds's "System of Medicine," vol. iii. p. 475, 1871.

‡ "Der Mechanismus der Respiration und Circulation." Berlin, 1845.

The second is atrophy, in consequence of which the partitions between neighbouring air spaces give way, and larger cavities are formed by the union of two or more smaller ones. These are the "permanence-securing" causes of Jenner.

Emphysema is usually a secondary affection, of which the antecedents are bronchitis, hooping-cough, and other diseases producing dyspnœa. In some cases, however, the emphysema seems to be the original complaint; in others it appears to be hereditary, and intemperance sometimes has been assigned as a cause. For such cases, if they really exist, as well as for cases of universal emphysema of both lungs, quoted by Waters, the mechanical explanation is insufficient, and we must suppose some primary alteration in the lung tissue.

In horses emphysema is a very common disease, and constitutes the vice termed "broken wind," which veterinary surgeons state to be chiefly due to overworking after a full meal of green meat. Veterinary surgeons have observed that it is hereditary in horses,* which tends to confirm the like remark made by Dr. Budd, Louis, Hasse, and others,† as applied to man. Dilatation of the bronchi is a pathological condition frequently associated with vesicular emphysema, and may be attributed to the same cause.

Hypertrophy of the Lung.—There is a consecutive condition of the lungs which may be mistaken for emphysema. Thus, after a long-standing disease of one lung impairing its functions and diminishing its capacity, we find its fellow taking on a vicarious action, and expanding so much as to displace adjoining viscera; a point of importance in forming diagnoses of diseased states of the thoracic contents. On opening this side of the thorax the lung may appear too large for its cavity, and induce the assumption of an emphysematous condition. The history of the case, the examination of the lung, and the shrunken, contracted, bound-down, and atrophied condition of its fellow, will determine the real nature of the case. Whether in this instance there is an actual new formation of pulmonary vesicles, it is difficult to ascertain; Rokitansky is of opinion that such an hypertrophy of the lungs is due to a dilatation of the air-cells, with a simultaneous augmentation of their tissues; that it does not consist in an increase in the number of the air cells, but in their dilatation, the increased thickness of their walls, the enlarged calibre of their walls, and in the development of their vessels. There can be no doubt theoretically that the changes that take place must tend to increase the powers of aëration of one portion of the breathing apparatus; but an increased thickness in the walls of the air vesicles can scarcely facilitate oxygenation, unless it is by a reduplication of the basement membrane into the breathing cavity, so as to afford a larger surface for the capillaries to ramify upon. The special circumstances under which the secondary enlargement and contraction of the lungs take place, will be a subject for further consideration at a subsequent page, when we come to discuss the pathological states of the pleura.

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* See Mr. Youatt's work on the "Horse:" art. "Broken Wind."

† "Med.-Chir. Trans.," vol. xxiii. p. 37.

ATELECTASIS PULMONUM.

Diametrically opposed to the condition which we have just considered, is one which is peculiar to early life, and frequently has been confounded with the results of pneumonia. It consists in the permanence of the fœtal condition of the lungs; the vesicular structure either not being properly developed, or the infant not possessing sufficient force to expand the thorax, and cause the dilatation of the breathing apparatus. It was first observed, and duly described under the name of atelectasis pulmonum,[*] by Professor Jörg;[†] who found that it occurred chiefly in full-grown infants of very feeble powers, or in such as had been born prematurely, and were, therefore, not in a condition to dispense with the placental circulation. The inferior portions of the lungs are most liable to present this state; it occurs in patches, which offer a darkened colour, do not crepitate on compression, and present a smooth surface on section. The affected part sinks in water. The part or lobule that has not undergone the due expansion, is below the level of the surrounding dilated lung tissue. Atelectasis is not necessarily fatal; it is probable that in many instances it is entirely overcome, as the child acquires vigour, or that while a few lobules remain in a permanently contracted state, the remainder of the lung suffices for the purposes of aëration. We frequently, in adults, meet with puckerings of the surface of the lung without any trace of inflammation, which may perhaps be set down to this congenital state of the tissues; or possibly the small nodules of fibroid or calcareous matter, which we often find equally without appreciable recent lesion to which they could be referred, may be due to the same cause, as we know them to favour parts in which there is an arrest of development. Dr. West observes, that if air be blown into a lung, some lobules of which are not duly expanded, it will permeate the collapsed air-tubes; the pulmonary vesicles will by degrees become distended, and the solid lobules rise to a level with the rest of the lung, acquiring the same colour and consistence, and, like other parts of the organ, will float in water. A single inflation, however, is by no means sufficient to render this change permanent; but the moment the tube is withdrawn the air will escape, and the lobules recently distended will again collapse and sink below the rest of the lung; their colour, too, will become dark, though less so than before. In conjunction with imperfect expansion of the lung tissue, we invariably find the foramen ovale and the ductus Bctalli still open. The pathology of the affection, which we have attributed mainly to a mechanical defect, has been explained by French writers, who have termed it carnification, as the result of pneumonia; but although pneumonia may supervene in an atelectatic lung, the characters of the two diseases are sufficiently distinct to be discriminated on a careful examination.

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[*] Etymology,—ἀτελὴς, imperfect; ἔκτασις, expansion.

[†] "De Pulmonum Vitio Organico," &c. Lips. 1832; and "Die Fötuslunge im Gebornen Kinde." Grimma, 1835.

Acquired Atelectasis or Collapse.—Another form of atelectasis, which Bailly and Legendre first pointed out, and which has been well illustrated by Dr. West, is that which occurs after respiration has once been fairly established, and is the result of an interference with the mechanism of respiration. It is this form more particularly which Valleix, and Rilliet and Barthez, under the term of carnification, have attributed to lobular pneumonia; the affection being limited to a single lobule or to a cluster, forming a hard, compact mass, surrounded by the normal tissue. When the affected part is inflated the vesicles distend, and thus show that there is no inflammatory effusion. Since this change is commonly found along with lobular pneumonia, and the latter disease is said to become developed more especially in the collapsed portions of lung, the distinction is sometimes difficult. In a child, whose case is related by Dr. West, there was no evidence of disease until the age of nine months, although she had not thriven well, and had become pigeon-breasted. She then lost flesh rapidly, and began to cough, without having had any previous catarrh. Her case seemed to be one of bronchial phthisis. Four days before death she became suddenly oppressed, and the cough more severe; the dyspnœa increased, while the cough became less frequent. A few hours before death the lips were quite livid, she breathed from eighty to eighty-six times a minute; the abdominal muscles acting most violently, but the chest being scarcely at all expanded. No tubercle was found in any organ after death, but large portions of both lungs presented the undilated condition, which disappeared entirely on inflation; the bronchi were pale, and contained very little mucus; the right side of the heart was greatly distended with coagulated blood, which its thin, pale, and flaccid substance had evidently been unequal to propel with the requisite vigour.

This condition of parts of the lungs is not uncommon in children of feeble muscular power, who suffer from acute or chronic bronchitis; but especially in those affected with rickets. It may be attributed partly to insufficient expansion; partly to the blocking up of bronchial tubes, and, in addition to these causes, to the permanent diminution of the capacity of the chest, produced by rickety changes in the skeleton. Such portions may be recognized during life by the absence of respiratory sounds and partial dulness; after death by the same appearances as those seen in congenital atelectasis, from which indeed it cannot be distinguished. Acute general collapse of the lungs, with drawing-in of the ribs, sometimes occurs in severe bronchitis of rickety children, and is rapidly fatal.

Carnification.—Compression of the adult lung by fluid in the pleura, by tumours, by an hypertrophied heart, or other causes, gives rise to a condition analogous to infantile atelectasis, known as carnification. The collapse of the air-cells is in fact quite as complete and the lung equally deprived of air; but as the pressure from being external acts more uniformly, the change extends over larger portions, and exhibits a gradually increasing condensation. In early stages the carnified portion is purplish, full of blood and tough, but easily inflated from the trachea. Afterwards it becomes bloodless, slate-coloured, very dense, or almost like india-rubber, and is no longer susceptible of inflation. Partial changes of the same kind are also in adults attributed to bronchitis.

Results similar to those of compression may follow from the diminution in the size of the chest consequent on spinal curvature, and we meet with an analogous condition in advanced age—the apices of the lungs being converted into a dense melanotic mass, in which we are unable to trace tubercular deposit, while there is an obliteration of the vesicular structure, and apparently also of the blood-vessels. The tissues present a viscid, tenacious mass, deprived of all air. Under the microscope, we see an almost homogeneous membrane, through which the carbonaceous deposit is scattered irregularly, with only here and there a trace of the circular fibres of the lung. In the cases which have fallen under our own observation, there was a thickened pleura forming a cap over the apex, which must, by its compression, have contributed much to the obliteration: whether any pneumonia had aided in producing the result is doubtful; our impression certainly is against this view, the excessive deposit of carbonaceous matter associated with the large pleuritic exudation in the confined post-clavicular region of the thorax appearing adequate in itself to account for the effect produced.

ŒDEMA PULMONUM.

Before investigating the inflammatory conditions of the lungs, we are required to devote some consideration to a morbid state which we frequently meet with in the dead body, and which, since the attention of the profession was first especially directed to it by the researches of Laennec,* has been known by the term of Œdema Pulmonum. It consists, as its name implies, in a serous infiltration of the interstitial portion of the pulmonary parenchyma. It causes a puffiness of the organ, which pits more or less on pressure, has lost its natural crepitant sensation, and does not collapse when the thorax is opened. The œdematous lung is characterized by pallor and anæmia, and when cut into discharges an abundance of clear, limpid serum, in which the comparative absence of air-bubbles is characteristic. Pulmonary œdema occurs in connection with, and as a result of, a great variety of debilitating diseases. It very commonly supervenes immediately before death; and has been attributed to the extinction of the nervous power of the vagi, in consequence of the experiments of Müller, which have shown that the fatal effect of dividing these nerves in the neck is mainly due to the infiltration of the lungs and air-passages with serum. Both lungs are generally affected to the same extent, nor is the œdema necessarily confined to the posterior portions, even where it is only partial. The most rapid and serious form of œdema is that which accompanies general dropsy, especially that caused by disease of the kidneys. A peculiar fact remarked by Hasse is, that where, in general dropsy which proves fatal, the one lung is found universally adherent to the pleura, and the other not, the former is œdematous, and the latter compressed by hydrothorax. Laennec states that pneumonia induces a great proclivity to the production of pulmonary œdema during the period of convalescence ; this may have been partly due to the excessive depletion formerly in vogue, for pulmonary œdema appears to occur so frequently, and so much in the ratio of the general anæmia of the individual, that we see no reason for assuming a special tendency in one disease to produce it. The lax texture of the lungs, like that of the superficial cellular tissue, necessarily favours a serous effusion under such circumstances. A collateral œdema of the unconsolidated lung often accompanies unilateral pneumonia.

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* "De l'Auscultation Médiate,' vol. i. p. 349.

PULMONARY CONGESTION.

The spongy texture of the lungs, coupled with the fact that these organs contain a larger quantity of blood, in proportion to their size, than any other organ of the body, renders them peculiarly liable to the various forms of congestion—a tendency which is enhanced by the relation existing between the pulmonary and systemic circulation. There are doubtless very intense and possibly fatal forms of congestion which are not recognizable after death. It is within our experience, verified by post-mortem examination, that death may occur with extreme rapidity from congestion alone. Sudden and intense hyperæmia was formerly called vascular pulmonary apoplexy, a term now applied to hæmorrhagic infarctus. Most of the various causes of death, while inducing an arrest of the circulation, give rise to an accumulation of blood in the organs of respiration. Nysten's experiments have shown that the contractile power of the right side of the heart continues long after the irritability of the left side is extinguished ; and the effect of maintaining artificial respiration in cases in which death from a lesion of the nervous powers is to be apprehended, further demonstrates the great share taken by the lungs in the production of death. The elasticity of the arteries also exercises some influence in producing an engorgement of the lungs, at the moment of, and immediately after, death, by propelling their contents into the venous system, and thus overcharging the right side of the heart. Nor must we forget that, in long standing debilitating disease, whether a specific fever or an adynamic condition, resulting from other disorganizing processes, respiration is carried on with little vigour, while the muscular tone is reduced to a low ebb, so that both causes conspire to retain the blood in the pulmonary tissues. Hence in estimating the pathological changes in the lungs connected with the actual disease to which death has been attributed, we must be very careful in distinguishing between the effects of dying and death itself, the secondary products of debility and dissolution, from the changes attributable to active disease. Both, however, often pass imperceptibly into one another, with gradations, which only confirm the view that disease is, in itself, incipient death. And though we may lay down rigid classifications of the modes of dissolution, and we may occasionally meet with types corresponding to our scientific arrangement, still, as Dr. Williams observes, in his "Principles of Medicine,"—"In the slower dissolution by which diseases generally prove fatal, all functions and structures are more or less involved, and life in all is dwindled down to so slight a thread, that, when it breaks in one, others scarcely retain it long enough to enable us to say that death begins distinctly in any part." Whether we can trace the death to asthenia or apnœa, coma or paralysis, the prevailing effect is to induce those symptoms to which we have above alluded, in the lesser circulation.

*Hypostatic Congestion or Hypostasis.**—The form of congestion produced by languid circulation during life, that which is induced in the state immediately preceding death, and the accumulation of blood in certain parts of the lungs after death, are practically indistinguishable, and are denoted by the common name of hypostatic congestion. This congestion is most liable to affect the posterior and inferior portions of lungs; after death, as in the debility resulting from disease, the blood follows the physical law of gravitation, and sinks to the lowest point it can gain. If there be no concomitant inflammatory changes, the congested portion presents a dark red colour, and, though firmer than the more bloodless anterior part, still crepitates under the finger, and floats in water. The colour is almost uniform, and the line of definition between the congested and the non-congested portion is tolerably clear. The pleural surface of the engorged portion presents a corresponding violet tint, which sometimes is more or less circumscribed at single points. The depth of the colour varies somewhat in different diseases; and in very anæmic cases, especially in those associated with general dropsy, there is more or less serous effusion with the sanguineous congestion. The extent to which hypostatic congestion occurs after death depends partly on the time which has elapsed since death, being greater as this is longer; and also not less decidedly upon the fluidity of the blood: since only blood which is uncoagulated is free to obey the laws of gravitation. In a medico-legal point of view, congestion of the lungs may become a question of life and death; thus, in the trial of Mr. Kirwan, in Dublin, the conclusion that his wife's death was due to violence, which has since been shown to be erroneous by the highest authority in medical jurisprudence in this country, Dr. Taylor,* was based mainly upon the fact of the lungs being congested posteriorly. This was the main fact upon which the medical witness, Dr. Hatchett, relied, in proof of death having been brought about by drowning; we know that it may be the result of post-mortem changes, and, as Dr. Taylor observes, "it is not of the least value as medical evidence of drowning, unless observed soon after death, and unless attended with other appearances, which, upon the assumption of death by drowning, or by some other form of asphyxia, ought always to accompany it."

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* ὑπόστασις (in Hippocrates)=sediment, or a sinking to the bottom. Thus hypostatic means *sedimentary*.

It is doubtful whether there are any means of determining whether hypostatic congestion has occurred after death or within a few days of dissolution. We know that in full vigour the blood is not disobedient to the laws of gravitation, as we may easily ascertain by allowing our arm to hang down and then raising it into a vertical position, or by elevating our feet above our head; therefore it is not surprising that, in the recumbent position, as the powers of life fail, the blood should gravitate to the posterior portion of the lungs. If the congestion is confined to one lung, or to the anterior parts of the lung, we may safely attribute it to morbid processes; and if there are any other traces of inflammatory action, to which we shall advert further on, we may equally set down the congestion to a pathological cause.

In the words of Förster, the post-mortem appearances can only in such cases be rightly understood by a knowledge of the physical signs of disease detected during life.

Hypostatic congestion is closely allied to the disease which has been termed "pneumonie des agonisans," by Laennec, and has been fully described by Mr. Erichsen † as the congestive pneumonia to which the majority of deaths following capital operations are due. It may also, if it exist long, be of course a cause of inflammation, and may thus pass into the hypostatic pneumonia, so frequent in adynamic diseases, such as enteric fever.

Chronic Pulmonary Congestion.—When any obstacle occurs to the passage of blood through the left side of the heart, whether from obstruction of the auriculo-ventricular orifice, incompetence of the mitral valve, feebleness of contraction of the muscular substance, or more remotely, obstruction or regurgitation at the aortic valves; in all these cases the lungs will be chronically over full of blood, and its capillaries exposed to undue pressure. The changes produced in the lungs by these conditions when they become chronic are very important, but though they have received various names as splenization and brown induration, they are not always clearly distinguished.

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* "Dublin Quarterly Journal," Feb. 1853.

† "Medico-Chirurgical Trans.," vol. xxvi. p. 29.

In early stages we find the lungs bulky and heavy, the substance deep red from vascular fulness, dense, containing little air, and more or less indurated, tough and difficult to tear. In this condition the term splenization has especially been applied;* but this word has been used in so many senses that it is perhaps better avoided. In the later stages of cardiac congestion of the lung, the induration and toughness notably increase; the colour is rather rusty brown, or more rarely slaty, as may be seen when the blood in the vessels is washed away. Much brown or dirty serum may often be squeezed out.

This is the condition described by the German pathologists as *brown induration.*

The histology of this condition is very interesting. On making a thin section from a hardened specimen, it is at once obvious that the air cells are unusually small in diameter. This diminution might naturally be ascribed to thickening or hypertrophy of the walls, but (as admirably shown by Rindfleisch) it really depends upon the projection into the cavity of the immensely distended pulmonary capillaries. At the same time the air vesicles contain numerous catarrhal cells, some epithelial in shape, but most resembling leucocytes or mucus corpuscles. All these are very highly charged with pigment, and the same is found in considerable quantity in the alveolar walls. It is impossible to deny, at least in the later stages, the simultaneous occurrence of fibrous hypertrophy, and it has lately been shown that there is an increase in the number of smooth muscle fibres. These anatomical changes suggest at once two factors in the dyspnœa of cardiac disease. In the first place, the air cells being diminished in size, the respiratory surface is also diminished; and, in the second place, the capillaries will be filled rather with arterial than with venous blood; the former being as little suited for the functions of the lung, as the latter for the nourishment of the tissues.

PULMONARY HÆMORRHAGE.

Diffuse hæmorrhage into the parenchyma of the lung may, it is probable, sometimes occur simply in consequence of extreme engorgement, as in the hyperæmia following on heart disease. When the lung is in a condition of intense, almost black splenization, the additional redness caused by a little hæmorrhage will be hardly distinguishable. More commonly distinct masses of hæmorrhage are seen, which will be presently described. Bleeding from a larger vessel, sufficient to cause laceration of the lung tissue, is not very common, and the conditions producing it, except aneurism, are not well understood. To this form, as resembling cerebral apoplexy, the term pulmonary apoplexy might most reasonably be applied, especially as it is usually fatal. The name has, however, by custom been assigned to a different condition. Hæmorrhage, more or less extensive, also occurs in pulmonary phthisis; of which more will be said in the proper place.

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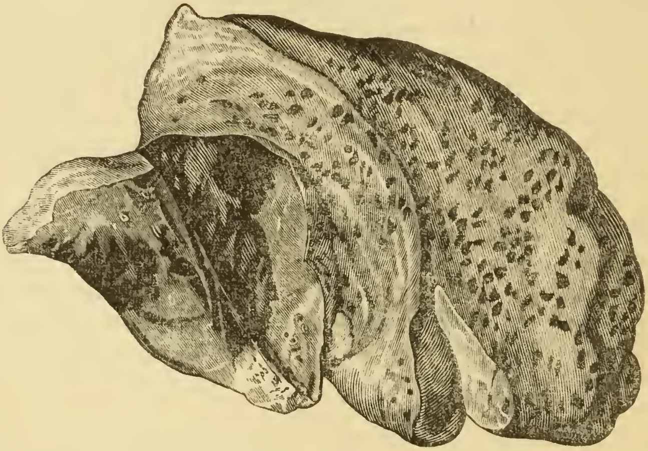
* Wilks: "Lectures on Pathological Anatomy," 1859, p. 223.

The commonest form of hæmorrhage is that which occurs in circumscribed masses. and is always associated with disease of the heart, or embolism—viz., the hæmorrhagic infarctus, or pulmonary apoplexy.

Hæmorrhagic Infarctus, or Pulmonary Apoplexy.—This form of hæmorrhage is distinct from that which occurs from the bronchial mucous membranes, either owing to an adynamic state of the blood or to active congestion, both in the symptoms it produces during life, as well as in the post-mortem appearances. The seat of pulmonary apoplexy is the parenchyma of the lung, blood being effused into the infundibula and into the air vesicles themselves, but not usually spreading so far as into the bronchi. The apoplectic spot may be felt before the lung is cut into as a globular or wedge-shaped mass, of greater density than the surrounding tissue, and, if near the surface, its darker colour also attracts attention, while there is usually inflammation and thickening of the pleural surface over it. On section, we find, if the hæmorrhage be recent, a dark red, almost black, homogeneous, circumscribed spot, varying in size from a pin's head to an orange, of the appearance and consistency of damson cheese, bounded by tissue, which is

Fig. 103.



Pulmonary apoplexy, occurring in a man aged fifty-three. There were several apoplectic masses, exhibiting a deep purple, almost black hue, and causing a homogeneous solid appearance of the part affected, as shown in the section.

comparatively healthy, both in colour and consistency. The only interruption to the uniform colour that is met with is that caused by the divided bronchules, which are less dark than the surrounding parts; the more recent the hæmorrhage, the more defined the outline. The breathing capacity of the part is entirely destroyed; it contains no air, and when scraped, only yields a dark, thick, bloody fluid, in which the microscope detects nothing but blood corpuscles and some pulmonary débris. If the margins of the clot be scraped and examined, we may find exudation corpuscles, varying in size from $\frac{3\text{-}7}{5000}$ of an inch, showing that some inflammation is going on.

The most important point to be noticed with respect to these hæmorrhagic masses, is that the whole arterial and capillary vascular system of the part is distended with stagnant and coagulated blood. The capillaries are in the condition of thrombosis, and the branch of the pulmonary artery distributed to the part is always found also blocked with a clot. This clot usually fills the vessel completely, ends abruptly towards the heart, but may be traced in the other direction into all the smaller branches. The surface is usually white and granular, like that of a clot which has sojourned long in a vessel, and the centre is sometimes crumbling, decolorized, or even softened. Very often the clot is heterogeneous in its composition, appearing to be made up of masses of different date. There is little doubt that the condition of the hæmorrhagic block is connected with the blocking up of the arterial branch; for, as previously shown (p. 397), the plugging of a terminal artery and distension of the capillaries (which are supplied by regurgitation from the veins) cause first great fulness, and ultimately hæmorrhage. It is only occasionally, and that in cases where a very large arterial branch is stopped up, that gangrene occurs. It has been supposed that in such cases the bronchial artery also is obstructed; but this cannot be proved.

While it is generally admitted that the stopping up of the blood current in the artery will produce stagnation and thrombosis in the capillaries, opinions differ as to the immediate cause of the blocking of the artery. Some regard it as a thrombosis, others as an embolism. In support of both views is urged the fact that coagulation in branches of the pulmonary artery is always accompanied by coagulation elsewhere, either in the heart, especially in the right auricle, or in the veins, or both. It is urged by the advocates of thrombosis that the same causes which effect coagulation in the heart or in the veins may effect it also in the pulmonary artery. Those who attribute the phenomena to embolism, believe that fragments of clot are washed from the right side of the heart, or from some vein, into the pulmonary circulation. It is extremely probable that in many cases the two processes are combined.

From what has been said, it is clear that arrest of circulation in a part of the lung, from whatever cause, may produce hæmorrhagic infarction. Practically the condition is hardly met with, except in cases of heart disease, where the pulmonary circulation is impeded. Formerly hæmorrhage was explained as a simple consequence of excessive pressure; but this does not account for the occurrence of isolated blocks, for their peculiar shape, or for the obstruction of the vessels. Heart disease then appears to act chiefly as a cause predisposing to coagulation of blood in the vessels. Also (if the explanation we have given be correct), by increasing the tension in the pulmonary veins.

Changes in Hæmorrhagic Blocks.—A block of pulmonary apoplexy once formed, soon begins to undergo a process of evolution. The central parts become paler, and ultimately white, while at the same time they are drier and more friable. Next softening sets in, and the mass may liquefy into something very like pus in external appearance. No increase in the number of leucocytes or lymphatic cells is, however, observed, the whitish mass, whether solid or liquefied, showing under the microscope chiefly obscure granular matter and fat, with only scattered corpuscles. These changes are not due to the re-establishment of circulation or to inflammation, but to a sort of necrosis, or "necrobiotic decay," as in tubercle. In the outer parts of the block, multiplication of elements and inflammation take place, resulting in the formation of fibrous tissue. Thus a zone of extremely hyperæmic substance will be found surrounding the softened and decoloured central parts. At the same time there is always some inflammation of the pleura, usually with formation of false membranes of lymph. Finally, all the central part may be absorbed, and all that remains of the block will be a cicatrix of fibrous tissue, producing a depression on the surface of the lung.

CHAPTER XXXII.

PNEUMONIA.

Several morbid states have been called by this name, but it more especially belongs to that form of inflammation in which the air-cells are filled with fibrinous exudation and leucocytes. This is the disease distinguished as *lobar*, *croupous*, or *fibrinous* pneumonia.

LOBAR PNEUMONIA.

This inflammation of the pulmonary tissue is commonly assumed to present three stages, which we may trace in regular succession in the patient, or which we find co-existing at various portions of the lungs in the same dead subject. The first stage, that of congestion or engorgement, we have already considered; its situation, its effect upon the cohesion of the tissues, the co-existence of other inflammatory changes, and the history of the case must assist us in determining its character, though it is often difficult to be certain of its nature. The general effect of acute inflammation in altering the cohesion of the tissues, is a point of considerable importance; when it particularly affects, as Sir Robert Carswell has pointed out, the uniting connective tissue element, it may thus demonstrate the previous existence of inflammation, though the redness and vascularity have disappeared, or but faintly mark the degree of alteration which the disease has effected in the process of nutrition. This general law is compatible with the observation that the second stage of pneumonia, or hepatization, is accompanied by a state of solidification; for, as the author named remarks, though the tissues feel harder than natural when compressed, the diminution of cohesion which has taken place between their anatomical elements, is rendered conspicuous by the facility with which they are penetrated, broken down, or crushed.

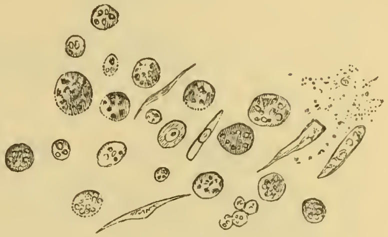
In doubtful cases the microscope would aid us by determining the presence or absence, in the congested portion, of exudation corpuscles, which we find where the naked eye fails in distinguishing the existence of inflammation. The confines of the first and second stages merge into one another, and are often difficult to define. In the second, the stasis of the blood becomes more marked, the specific gravity of the pulmonary tissue increases, the overcharged vessels relieve themselves by fibrinous exudation, and by slight hæmorrhage into the air cells and infundibula ; the latter mingling with the bronchial secretions, gives rise to the pathognomonic rust-coloured expectoration of pneumonia. This stage has received the name of hepatization, owing to the increased density of the parenchyma causing the affected portion of the lungs to resemble a piece of liver.

The colour of the affected part is of a dark red, which is more or less venous or dusky, in proportion to the type of the inflammation ; the crepitant character of the tissue is fast disappearing ; the lung, on section, has lost that light, spongy appearance peculiar to it in health, and but little frothy red fluid exudes from it. On the pleural surface, instead of the slate-coloured, marbly hue of the normal state, we find a more uniform, dusky red colour, scarcely broken by the interlobular septa. At this period, as Gendrin first pointed out, repeated washing and continued maceration fail to restore the natural colour of the tissues, which they recover, under such a process, if the redness is due merely to congestion. Before that exudation and general infiltration have taken place, which constitute the succeeding stage of pneumonia, we find that, on breaking up a portion of hepatized lung, the surface is studded with small pinky granulations, which are identical with the pulmonary granulations of Bayle. This author, and some of his successors, looked upon them as the first stage of tubercular disease ; but, as Andral has satisfactorily shown, they are a product of inflammation, being, in fact, the solid masses contained in the alveoli and terminal air passages. On scraping the cut surface we may sometimes succeed in obtaining pretty accurate casts from the interior of these structures.

Though the specific gravity of the hepatized lung is considerably increased, the dimensions of the organ are not necessarily augmented ; occasionally, however, the organ is shown to be enlarged by the indentations left on its surface by the ribs.

The different degrees of congestion of the inflamed portion produce a mottling of the surface ; the congestion itself may be circumscribed with a definite line, or it fades off gradually into the healthy tissue. As the second stage progresses, marbling of a different kind occurs, which is due to the gradual disappearance of the colouring particles of the blood, the absorption of the blood itself, and the substitution of a fibrinous deposit, or pus. We now enter into the third stage of pneumonia, or that of grey hepatization. The term well denotes the appearance of the affected portion ; it is entirely consolidated and deprived of all air, it presents a greyish or greyish-yellow colour, which is only varied by the almost linear remains of the compressed bronchules, and the pigmentary matter scattered through the lung tissue. The general condensation of the tissues necessarily, also, involves the blood-vessels, and arrests and prevents the circulation; so that the further changes must be mainly due to extra-vascular metamorphoses. The parenchyma becomes gradually softer, and the more straw-coloured and paler its hue, the more fully the suppuration process is established, and the more friable the tissue becomes. A purulent fluid now exudes on pressure. At the commencement of this stage the air vesicles may be isolated in the shape of grey granulations, of a globular form, containing an opaque granular matter. The microscopic characters of grey hepatization at this period have erroneously been stated to be those of suppuration only; but true pus cells are by no means the predominant forms seen.

Fig. 104.



Microscopic characters of the contents of an air vesicle in grey hepatization, consisting of granular matter, pus corpuscles, exudation cells, and cylindrical epithelium.

When the third stage of pneumonia advances to a fusion of the inter-vesicular septa, and an entire breaking down of the tissues, all trace of the normal structures disappears, and we only find a confused mass of pulmonary débris, pus, and ichorous sanies. We then have to deal with genuine pulmonary abscess; but this mode of termination must be regarded as decidedly exceptional.

MINUTE CHANGES IN PNEUMONIA.

If fine sections be made from a portion of lung in the stage of red hepatization, which has been subjected to appropriate hardening processes, we find an appearance like that represented in Fig. 105. The capillaries are gorged with blood, the epithelium unaltered, the cavity of the air cells partially filled with a mass made up of fine fibrinous threads and a variable number of "exudation cells" or leucocytes, with a few red blood corpuscles. The fibrinous mass, though not in contact with the alveolar wall, is nevertheless attached to it by fibrinous prolongations, which pass out into neighbouring alveoli. The fibrinous threads are notably fine and transparent. This appearance is only seen when the hepatization is very recent, and we do not often obtain specimens just in this stage.*

There is no evidence in this stage of any new formation of cells in the lung. The masses which choke up the air cells seem to be composed of some coagulable material effused from the vessels, which forms the fibrinous network, and elements which also come from the blood. The presence of some red corpuscles explains the well-known rusty colour of the sputa in pneumonia.

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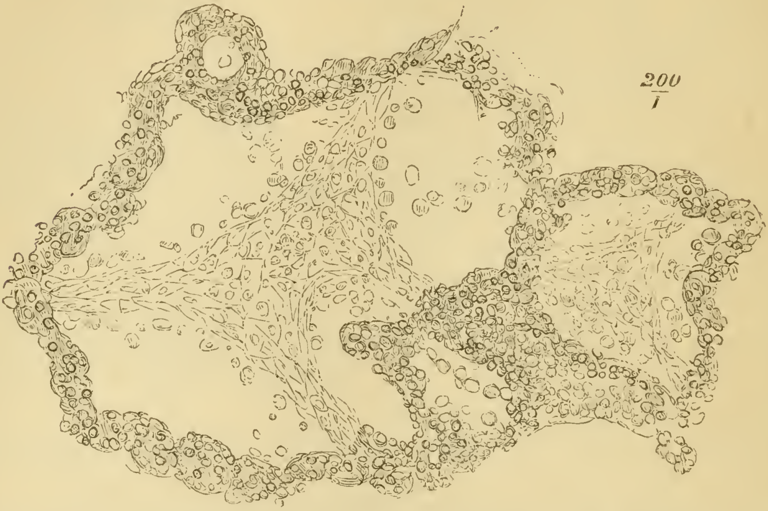
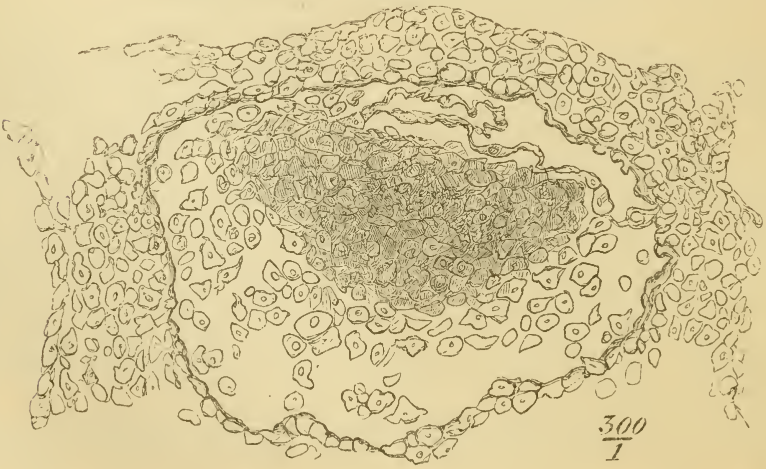
* In the lung from which Fig. 105 was copied there was some abnormal dilatation of the air cells, which perhaps exaggerates the space between the exudation mass and the walls.

Fig. 105.



Two alveoli of a lung in the first stage of pneumonia. The larger is somewhat abnormally dilated. Translucent masses of fibrin entangling numerous corpuscles are seen in the middle. The walls are unaltered, except that the capillaries are gorged with blood.

Fig. 106.



Alveolus from second stage of pneumonia. The opaque mass in the centre is unattached to the walls, and is surrounded by epithelial cells with other corpuscles. Similar epithelial cells are seen in large numbers covering the capillary wall.

At a later stage (Fig. 106), when the lung is already grey and granular, the appearance is somewhat different. We then get a much more opaque mass in the centre of the alveolus, with a more rounded outline, and surrounded by a large number of cells of irregular form, and apparently more or less flat and epithelial in character. Similar cells are seen covering very thickly the walls themselves. The capillaries in this stage appear generally empty after death, a fact which explains the *grey* colour of the lung, though it is not easy to conceive of them as other than very full during life. The opacity of the exudation mass shows that changes are going on in it—probably in part fatty degeneration—and its rounded shape shows that it is becoming detached from the wall on all sides, a difference which accounts for the granular appearance of this stage of pneumonia, and the ease with which the rough casts already mentioned are obtained. Furthermore, the abundant production of new cells of epithelial character shows one source of the new elements of pneumonia, and probably one factor in the loosening of the exudation mass from the walls. (This is what is called by Rindfleisch "catarrhal desquamation of the alveolar wall.")

In a later stage, when resolution is fairly in progress, the mass is still more granular and indistinct, while the number of new cells is immensely increased, and these are more "indifferent" in character, without the pronounced epithelial form. This constitutes "purulent infiltration," and is the process which fits the masses contained in the air cells for expectoration.

There are diversities, as might be expected, in the minute characters of the different varieties of pneumonia. When the disease occurs in the course of typhoid fever, and in other forms of what is called "low" pneumonia, the amount of fibrin is very small, and the alveoli are mainly filled with cells. Sometimes the fibrin appears to be in excess, which gives great hardness to the lung. This seems to occur chiefly in forms of pneumonia, which tend to become chronic.

DISTRIBUTION OF PNEUMONIA.

The parts most liable to idiopathic pneumonia are the inferior portions of the lungs, while the upper lobes and apices of the lungs are rarely affected with pneumonia, except in connection with tubercle. Louis states that he has constantly found pneumonia affecting the upper and anterior part of the lungs, without a trace of the disease existing posteriorly, to be tuberculous; and he lays it down as a rule that this localization of pneumonia may lead to the diagnosis of tubercular disease previously undiscovered. This is undoubtedly correct in the main, but as Sir Thomas Watson observes, it is probably exaggerated; and exceptions to the rule regarding pneumonia are met with, as well as in reference to the ordinary site of tubercle. Indeed, the numbers given by Andral would almost destroy the validity of the law altogether; for though they yield the preponderance to the lower lobes, they seem to show a much greater proclivity in the upper than they are commonly supposed to possess; in eighty-eight cases of pneumonia, he found it limited to the lower lobes in forty-seven; in thirty the upper lobes, and in eleven the entire organ was inflamed.

The general tendency of pneumonia is to spread from below upwards, and for this reason we commonly meet with the several stages in the same lung; the base to a greater or less extent presenting the grey hepatization—red hepatization affecting the adjoining portion next above the former—while the upper lobe offers more or less pneumonic congestion. The smaller bronchi of the affected part are not, as has been stated by several authors, invariably affected at the same time; we often see them meandering, as white rivulets, through the inflammatory mass; and they occasionally appear to possess a repellent power, and to form a line of demarcation between two parts that are unequally affected. A curious exception is mentioned by Rokitansky, in which, owing to bygone pleuritic effusion, the base of the right lung had not recovered its pristine elasticity, and where the entire lung was in a state of red hepatization, with the exception of the apex, the anterior margin, and the base, which latter is scarcely ever found exempt. It is equally exceptional to find pneumonia limited to the central portion of the lungs, though such cases undoubtedly occur; hence one of the most ordinary complications of the disease is with pleurisy; for which reason some authors prefer the compound term pleuro-pneumonia, to the separate names pleuritis and pneumonia.

According to the unanimous testimony of all observers, the right lung is more frequently affected than the left, while double pneumonia is less often met with than either; the analysis of 210 cases of pneumonia by Andral* yielded the following results:—

The right lung alone was the seat in	121
The left in	58
Both together in	25
The seat undetermined in	6
Total	210

The relation is somewhat, though not essentially, altered by the larger numbers collected by Sir John Forbes; he finds that in 1,131 cases—

The right lung was affected in	562
The left in	333
And both together in	236
Total	1,131

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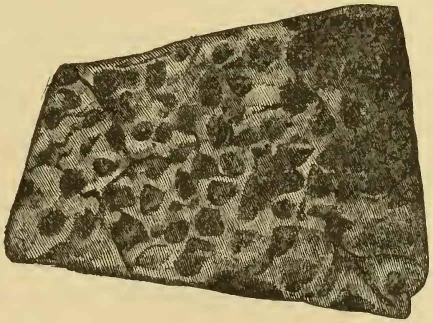
* "Clinique Médicale," vol. iii. p. 470.

FORMS OF PNEUMONIA.

The varieties of pneumonia which are spoken of by authors, as differing from ordinary acute pneumonia, are all characterized by presenting a more asthenic and less acute type, than the ordinary form which we have just discussed. The hypostatic pneumonia of Piorry, typhoid pneumonia, congestive pneumonia the bilious and the erysipelatous form of Riverius and older writers, the senile pneumonia described by Hourmann and Dechambre, are all forms of asthenic pneumonia, occurring in subjects debilitated by other diseases, in whom the lungs are attacked by a low, insidious form of inflammation. The congestion is of a more venous character, causing a dusky brownish violet tint; the physical law of gravitation exerts a strong influence upon the blood in the thorax, and the posterior portions are predominantly affected; the tissues are more friable and lacerable even in the first stages, and in the progress of the disease we fail to find that evidence of plastic inflammation which accompanies ordinary sthenic pneumonia. Much yet remains to be done with regard to determining the actual morbific agent in many of the forms of disease with which we have to deal; and when we shall have arrived at a correct appreciation of the constitutional tendencies, which induce the palpable manifestations of inflammatory action, we may hope to reconcile theory and practice more fully than has yet been done; we may then also explain why such various modes of treatment are successful in combating symptoms, which to our short-sighted vision indicate the same disease, though in reality they are due to totally different causes. Thus pneumonia, occurring in a system in which there is a predominance of lactic or lithic acid, would necessarily demand different remedies from one in which an alkaline or septic principle prevails.

Fig. 107.



Pleural surface of a portion of splenified lung, affected with typhoid pneumonia, from a female aged twenty-five, who died of typhoid fever. The lung closely resembled the spleen in consistency, was of a brownish red hue, interspersed with deep purple spots of an apoplectic character.

Hypostatic Pneumonia.—But to return to our legitimate sphere: the hypostatic form of pneumonia occurs in a variety of diseases in which the patient is confined to the recumbent posture, and in which the system is much debilitated. Under the name of congestive pneumonia, it has been described by Mr. Erichsen as the most fertile source of the fatal issue of capital operations, after the patient has survived the first shock, and therefore calls for the special attention of the operating surgeon.

In this form the congestion is by far the most prominent symptom; and though enough of vitality remains to give rise to some active symptoms, yet the debility of the patient, and of the organs of respiration, is so great as to prevent a reaction of a vigorous character. Mr. Erichsen* has analyzed sixty-two post-mortems of individuals who died after operations, and has set down as pneumonic only those cases in which either one lung alone was affected, or else in which some other palpable sign of inflammatory action was manifested in the cavity of the thorax, beyond a merely congested or softened condition of these organs, such as solidification of their tissue, whether hepatization or splenification of it, the effusion of recent lymph or serum into the pleural sacs, or marked evidences of inflammation of the bronchial mucous membrane. The result of his analysis is as follows:—There were twenty-eight in which there were evident signs of pneumonia; eleven in which the lungs presented the characters common to the first stage of pneumonia and passive congestion; nine where the lungs were diseased, but neither inflamed nor congested; and fourteen in which these organs were healthy, though many may have presented cadaveric congestion. A fact connected with this form of pneumonia, and pointed out by Mr. Erichsen, is, that while in ordinary sthenic pneumonia the right lung is most frequently affected, the left next, and lastly, both organs conjointly, here the two lungs are most frequently affected together. In the twenty-eight cases in which pneumonia was traced, the right lung alone was affected three times, the left lung alone also three times; and both lungs together, though not to the same degree, in twenty-two cases.

Typhoid Pneumonia.—In the pneumonia accompanying typhoid fever, congestion is equally a predominant symptom, and requires the more to be carefully watched during life, as the insensibility of the patient and the general torpor of the nervous system allow it to run its course without producing (in many cases) any symptoms of inflammatory action beyond those obtained by the stethoscope. Cough, expectoration, and dyspnœa may be absent, while we find extensive crepitation over a large extent of surface. In addition to the post-mortem appearances already detailed, we find the affected parts very soft, and the product is of a chiefly cellular character, without much fibrin, while there is no definite limitation to the disease. In some cases of atypic pneumonia, where we have reason to suspect an arrested secretion of bile in the liver, we find a genuine jaundice of other viscera, and then the frothy juice exuding from the cut surface of the pneumonic lung may present a yellow tinge, which has probably given rise to the term of bilious pneumonia.

Senile Pneumonia.—Again, in senile pneumonia,† the following varieties are described as belonging to the second stage; the lung, on incision, appears perfectly smooth and homogeneous, discharging a reddish, frothless serum, the inflammation occurring in patches, which are elastic, or soft; or, if granulations are present, they are much larger than in younger individuals. In the third stage a peculiarity has been occasionally observed by Hourmann and Dechambre, which Hasse confirms; it consists in the purulent matter being sharply defined in spots of from one to two lines in diameter, which prove to be the irregularly dilated air cells, often met with at intervals throughout the lungs of aged persons.

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* "Medico-Chirurg. Transact.," vol. xxvi. p. 29.

† Hasse's "Pathological Anatomy," p. 229.

The pneumonia, as we have described it, is the form which has received the name of lobar pneumonia as affecting entire lobes, in contradistinction to the lobular form of pneumonia, in which it is limited to individual lobules scattered through the healthy lung tissue. This, which is in fact a partial pneumonia, will now be discussed.

LOBULAR OR CATARRHAL PNEUMONIA—BRONCHO-PNEUMONIA.

Beside the form of pneumonia we have just described, there is another which differs both in its distribution, in the sequence of morbid changes of which it forms a part, in the time of life at which it occurs most frequently, and to some extent in its anatomical characters. Inflammation of the bronchi seldom extends, as we have said, into the lung parenchyma; and this extension is especially rare in adults; but in children it is not very uncommon. In such cases bronchitis may lead to the form of pneumonia we are now considering, which, at all events in the first place, affects single lobules or groups of lobules, instead of an entire lobe. The affection appears to consist in an actual extension to the alveolar epithelium of an inflammatory process similar to that already affecting the bronchial mucous surface; hence the term catarrhal pneumonia. Some regard it, however, as a mere blocking of the air cells with products of bronchial inflammation.

The lung usually appears solidified only in patches, the condition of which is not always absolutely to be distinguished from that of the whole lobe in lobar pneumonia. It shows, however, a greater tendency to become yellowish, dry, and crumbling, or sometimes the ramified arrangement of the terminal air passages, filled with yellowish masses, can be clearly made out. The most characteristic appearance is when we find, at the termination of the smallest bronchial divisions, spots varying from a pin's head to a pea in size, either of yellow granular material or of actual puriform matter.

This most characteristic appearance depends upon the anatomical structure of the lung, and is produced by the manner in which the morbid process is continued from the bronchial tubes into the groups of air cells; that is, by the fact that the air cells connected with a single terminal bronchial tube are simultaneously affected. The shape of a terminal bronchial tube, with its groups of air cells, is roughly like a bunch of grapes, or more closely like a racemose gland. This form is shown when a lung is only imperfectly expanded, either by incomplete respiration or by artificial inflation. It is also seen in artificial casts of the internal cavities, and is equally marked out by the disease we are now considering. When we see this typical arrangement of morbid change in a lung, we know that the disease is one which owes its distribution to the arrangement of the air passages, not to that of the blood or lymphatic vessels. It is, in fact, broncho-pneumonia; and this arrangement belongs both to the broncho-pneumonia we are now considering, and to the more serious form of it which constitutes one of the varieties of phthisis. In more advanced stages of either disease, when the small masses become confluent, this appearance is quite lost.

It often happens that either the part of the lung in which these masses occur, or else a closely contiguous portion, is in a state of collapse. This condition is, as we have said often, a consequence of obstruction of the air tubes by bronchitis; and such collapsed portions of lung appear very prone to pass into a catarrhal inflammation, so that transitional stages may be found between the two. The process of consolidation, though at first affecting single lobules or small portions, may soon spread over a large part or the whole of a lobe. The consolidated portions are pale and often of a pinkish colour, entirely airless and singularly dry, without showing precisely the granular appearance of lobar pneumonia. As the disease extends these portions become intermingled with healthy lung tissue in such a way as to produce a mottled or marbled condition.

When the consolidation spreads over a whole lobe or over both lobes, it may be difficult to distinguish from ordinary fibrinous lobar pneumonia, unless some portions remain in a less advanced condition.

In general, as we have stated, inflammation of the bronchi precedes this form of pneumonia, and the signs of this will accordingly be found along with those of alveolar inflammation. But it may happen that the bronchitis has passed away at the time of death, or even (according to Rindfleisch) it may be absent from the first.

Histology of Catarrhal Pneumonia.—The inflammatory process in the air cells which characterizes this disease is not only propagated from the mucous surface of the bronchial tubes, but also resembles it in character. Hence we do not find the fibrinous masses entangling corpuscles which occupy the air spaces in fibrinous or croupous pneumonia, but cells which are believed to be the result of a catarrhal inflammation of the alveolar epithelium, and have, in early stages, a more or less epithelial character. This character is, indeed, not recognizable in later stages when the new-formed cells present the uniform or "indifferent" character of inflammatory cells in general. In general no fibrin is to be seen, but, nevertheless, in a considerable number of cases of broncho-pneumonia some air cells at least will show some fibrinous threads among the inflammatory cells. The process may be very well traced in those parts of a phthisical lung where the morbid change is just beginning (see Fig. 110 *d*).

Here we see large flat cells detached from the walls and lying free in the cavity mixed with other products of inflammation. (In the specimen figured, blood corpuscles are seen, but these have no relation to the catarrhal condition, but are a sign of phthisis.)

This appearance is very rarely met with, and in general we find the air cells occupied with indifferent roundish corpuscles like those produced in inflammation of the bronchi. This fact has led some observers (as Buhl) to deny altogether the existence of a catarrhal inflammation of the alveoli, and to speak of these cells as the results of bronchial inflammation drawn into the air vesicles during inspiration. The gradual consolidation of the lung tissue they believe to be due to filling up with bronchial products. We must admit that the histology of ordinary cases of broncho-pneumonia shows nothing to contradict this view.

Consequences of Catarrhal Pneumonia.—This form of pneumonia is particularly fatal in childhood, and that description which often succeeds measles has acquired an especially ill-omened reputation. The fatal result in many cases depends on the gradual obstruction of the air cells by products which the expiratory efforts cannot remove, and is accelerated by the addition of collapse. But in other cases this inflammation runs on into a destructive process, in which masses of consolidated lung tissue soften and form cavities, in fact a kind of phthisis. This constitutes what has been called scrofulous pneumonia, or caseous pneumonia, or formerly tubercular infiltration, and often produces the clinical symptoms of "galloping consumption."

ABSCESS.

We have already seen that the formation of abscesses, such as we meet with in other organs and on the surface of the body, is a rare event in the lungs, notwithstanding the frequency with which inflammation attacks them. Yet a suppurative destruction of the tissue occasionally leads to this result, and we then find a cavity varying in size from that of a marble to that of an entire lobe, and presenting ragged parietes; it contains pulmonary dêbris and pus, which partially infiltrate the adjoining tissues, as there is no lining membrane; or the abscess may have discharged its contents, and we then only meet with the irregular excavations with jagged walls. Formerly the frequency of pulmonary abscess was considered to be much greater than we now know it to be, because tubercular cavities were confounded with this lesion. Now that the nature of both affections is better understood, the error is less likely to arise; when we are in doubt, the history of the case, the situation of the abscess, the condition of the surrounding parts, the presence or absence of tubercular deposits in the lungs, are points that will aid us in establishing a correct opinion.

It should, however, be remembered, that the distinction is, in fact, not always clear, but that abscess resulting from inflammation may, if it continue to extend by a species of ulceration, constitute, in fact, one of the forms of phthisis. When this is not the case, healing takes place by the gradual formation of a capsule of connective tissue round the abscess and its cicatrization. The pus is either completely absorbed, or else remains incapsulated as a yellow caseous mass which may become calcified. This is, accordingly, one source of the wasted cicatricial nodules which have been erroneously regarded as always due to obsolete tubercular disease. Another occasional result of pulmonary abscess is gangrene; and more rarely the pus opens into the pleura, producing empyema.

The form of abscess just described must be carefully distinguished from the multiple or metastatic abscesses which occur in pyæmia, and the distinction is in general not difficult.

SECONDARY OR PYÆMIC ABSCESSES.

These abscesses are called *secondary*, because they are invariably dependent upon some antecedent suppuration or other form of inflammation at a distance from the lungs. They have also been called *metastatic*, from the erroneous belief that pus was absorbed and removed from one part of the body to be deposited in another. For the same reason, they have been called secondary *deposits*. By some writers they have been described as a form of lobular pneumonia, and may be called simply *multiple* abscesses.

The appearances by which we recognize these abscesses when in a mature condition are, spots of yellow pus, varying in size from a pin's head to a walnut, generally situated near the surface of the organ and surrounded by a defined patch of deeply-congested tissue, which may present a colour approaching to black; beyond this, the parenchyma is in a healthy condition, or, at all events, in a state totally distinct from the circumscribed disease. The pleural surface over them is always inflamed. We generally find several of these abscesses in various parts of the lungs. The pulmonary tissue of the seat of the abscess may have entirely disappeared, or we may be yet able to squeeze out the pus, so as to show the normal structure.

In an earlier stage they bear some resemblance to the hæmorrhagic blocks or infarctus already described, being uniformly dark red and engorged, and the capillaries being, as in such infarctus, obstructed; sometimes, though not always, blocks may be found in the larger arterial branches leading to the part. They are often situated near the pleural surface, and, before they are altered by suppuration, may be more or less wedge-shaped; but neither of these characters is constant. The pyæmic blocks undergo rapid necrotic change in the centre, as do the hæmorrhagic; but they change much more rapidly and soon soften into a puriform liquid. Actual pus is not, however, so constantly produced as would be supposed from the naked eye appearance; the liquefied contents of the "abscess" consisting chiefly of granular débris, fatty molecules, with a certain number of granular leucocytes and other bodies presently to be mentioned. In later stages, when the inflammation is intense, actual pus is produced. Pyæmic abscesses are always associated with an intensely febrile state and other symptoms characteristic of "pyæmia," a condition which has already been discussed. The only point necessary to discuss here is the nature of the connection between the original disease and its secondary manifestations in the lungs. It has already been stated that the capillaries, and often arterial branches of these blocks, are stopped up; and the resemblance between the pyæmic blocks and hæmorrhagic infarctions has been pointed out. There seems every reason to believe that the obstruction of blood-vessels is the starting-point of the process. This obstruction is doubtless due in many cases to the impaction of small fragments of softened blood clot derived from some vein in the part which was the original source of infection; and such clots, are, as we have seen, in many cases found. Sometimes, however, there is no thrombosis of these veins discoverable; and we must still suppose it possible that local coagulation may be caused by the poison (whatever it be) which is contained in the blood in pyæmia. The material contained in the obstructed vessels is described by Dr. Bristowe as a "soft, pulpy, yellowish material," or else as something more approaching to ordinary coagulum; sometimes also "distinct pus"—that is to say, leucocytes too closely aggregated to be normal blood elements. Now this is precisely what is seen in softening clots from veins. It is not like the result of simple coagulation, being, if fibrine at all, fibrine which "has undergone changes requiring time for their production, and often clearly in advance of the changes which have taken place in the patches of diseased tissue." It has been recently made more than probable that peculiar low organisms (a species of Bacterium) are present in the blood in pyæmia, and are either themselves a poison, or the vehicles of poison. The relations of thrombosis and embolism just traced will then explain to us, not the origin of this poison, but the manner in which it is carried from the original source of disease to the lungs.

GANGRENE.

The most marked form which septic disease assumes in the lungs is presented in cases of gangrene; it is, as Sir Thomas Watson observes, very seldom the result of acute inflammation, and is almost as uncommon as the formation of true abscess. Its cause is not always clear. Dr. Stokes has published some cases, in all of which the patients had been habitual drunkards; the abuse of spirituous liquors does not, however, seem to exert a uniform influence in its production, for, in chronic alcoholism, as described by Dr. Huss,* this lesion has not been met with. It is also remarkable that ergotism, which has a peculiar tendency to induce superficial gangrene, is not accompanied by pulmonary sphacelus. The same applies to the analogous disease spedâlskhed,† or the Norwegian leprosy.

It presents two forms, the *diffuse* and the *circumscribed*. In the former, the lung tissue that is involved is broken up into shreds, which hang into a cavity filled with a fœtid putrilaginous, discoloured sanies, and through which the bronchi and vessels may yet be traced entire. The gangrenous portion presents a variety of hues, in the different shades of green, brown, and black. The surrounding parenchyma is infiltrated with ill-conditioned pus. An entire lobe, or even the greater portion of one lung, is found to be in this condition, though the upper lobe appears to be most prone to the affection. It appears sometimes to result from extension of circumscribed gangrene; but sometimes its origin is quite obscure. In circumscribed gangrene, which is more frequent than the former, we find one or more patches, varying in size and of irregular form, scattered through the lungs. There is much less tendency to involve the adjacent parts, and the course of the affection is more chronic than the former. Laennec describes the colour of the mortified portion as black with a greenish tinge—the texture as moister and more compact than that of the lung, and its aspect closely corresponding to an eschar, produced upon the cutaneous surface by nitrate of silver. The neighbouring pulmonary tissue is in a state of inflammatory congestion; and, after the sphacelated spot has become detached, a false membrane, of a greyish, dirty yellow colour is formed, which secretes an ill-conditioned pus. At times the membrane is formed even before the entire separation has been effected. If the gangrene involves the pleura, rupture and discharge into the pleural sac may ensue, or otherwise the bronchi may be the channels by which the evacuation is effected.

Circumscribed gangrene appears to be always dependent upon obstruction of the arterial branches supplying the part; to be, in fact, one of the terminations of embolic infarction. We do not know why gangrene is not a more frequent result of this affection; but it appears only to occur when the infarcted portion is considerable in size, and may perhaps be connected with simultaneous obstruction of the bronchial artery.

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* See Dr. Huss's work on the subject: "Alcoholismus Chronicus," &c. Stockholm, 1849.

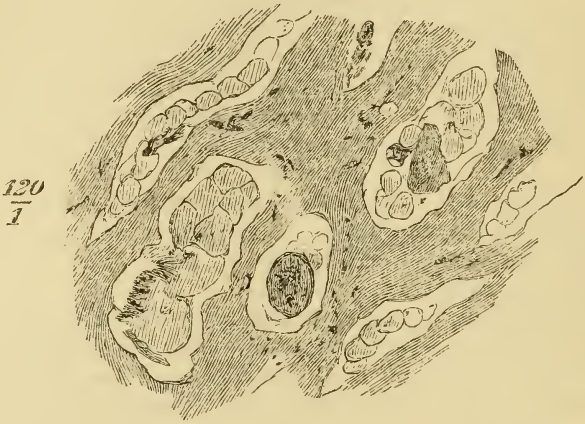
† Dr. Daniellsen and Boeckh, "Om Spedâlskhed," &c. Christiania, 1847.

CHRONIC PNEUMONIA.

The existence of chronic pneumonia has been disputed by some authors; but both at the bedside, as well as in the dead-house, we find cases to which no other name can be given, though, in the present state of our knowledge, it is often difficult to define the exact limits of the acute and chronic forms. Another difficulty which has yet to be removed, in regard to this subject is, to determine whether chronic pneumonia is essentially different from the destructive pneumonia which we shall describe among the forms of phthisis, and if so, whether there are any marked characters by which in the dead body, where we only have to consider it at present, it can be recognized.

The chief feature in chronic pneumonia, which we find equally dwelt upon by almost all observers, is the hypertrophy of the inter-alveolar tissues. Rokitansky, in contradistinction to his croupous form of pneumonia, gives the name of interstitial pneumonia to a second form, which he regards as identical with the chronic form of other pathologists, as for instance, Andral and Hasse. Rokitansky describes the inflammation as commencing between the pulmonary lobules and the smaller groups of air cells, and originating in hypertrophy of the connective tissue. A similar condition was first described by Corrigan as cirrhosis of the lung, and has also been called fibroid degeneration, or induration. The lung is usually much diminished in size, and the corresponding contraction is seen in the external wall of the chest. The pleura is greatly thickened, as are the interlobar and interlobular septa, so that thick fibrous bands intersect the lung, and divide it into irregular compartments of various size. The surface shows corresponding irregularity and nodular appearance, which suggested the name of cirrhosis, from a certain similarity to the appearance of the liver in that disease. The parenchyma of the lung is of a dark grey colour, with much pigment, and is itself indurated. The bronchial tubes are dilated, and sometimes cavities, with smooth internal surface, are formed partly, or wholly, by the enlarged tubes. They contain purulent mucus, often very offensive and gangrenous. This condition usually affects one lung only, and sometimes only one lobe. The above description applies to the most advanced stages of the disease. Early stages show little more than induration, and are not always easily recognized with the naked eye, though microscopic examination may show the characteristic thickening of the alveolar walls. The histology of a lung in this condition is represented in Fig. 108. The walls are seen greatly thickened, and containing pigment granules, while the air cells are filled with large irregular masses resembling mucus corpuscles, but often of abnormal size, as if several were fused together, and also containing pigment. It is not easy to say whether these bodies are produced where they are found, or have entered the air cells from the bronchial tubes.

Fig. 108.



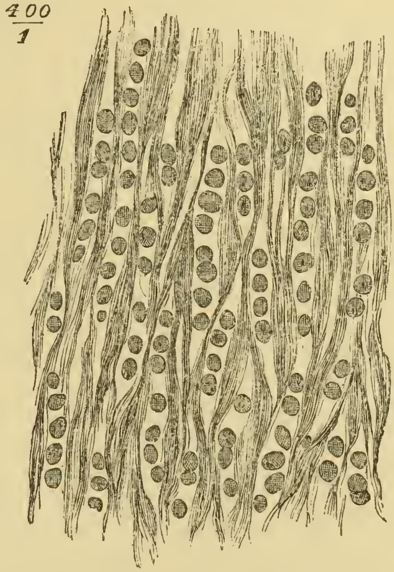
Section of an indurated lung affected with chronic pneumonia, showing the walls of the alveoli very greatly thickened, and the cavities filled with mucous corpuscles and larger masses containing pigment.

We do not, however, always find that chronic pneumonia consists in changes of this kind. It may, on the other hand, resemble acute lobar pneumonia in having the air cells filled with fibrinous exudation, and the only peculiarity observable in the exudation is the preponderance of tough, opaque granular fibrin and the small number of cells. In other cases we have found a remarkably luxuriant development of epithelial structures, which have quite filled the alveoli. Induration and contraction, however, in the end depend on the formation of a fibroid (now called lymphadenoid tissue) and gradual obliteration or replacement of the pulmonary tissue by it. This tissue is represented in Fig. 109. It varies in different cases in the proportions of the lymphoid cells and intercellular substance, but no doubt, with time, the former waste and the latter increases. Precisely the same structure is found in many parts of a phthisical lung.

Another result of chronic pneumonia is the formation of cavities beside and beyond the bronchial dilatations already spoken of. They are often lined by a very smooth membrane, but this is formed in course of time, and is only a sign that the cavity has existed a long time. Perforation of the pleura, pleurisy and empyema may result. Chronic pneumonia may, on the other hand, be the result of empyema when this has opened inwards into the pulmonary tissue.

The morbid process just described which includes fibroid induration and the formation of cavities, is evidently closely allied to ordinary phthisis, and has been often called fibroid phthisis. It differs, however, from tubercular or broncho-pneumonic phthisis in two respects at least. First, in the absence of tubercles and of caseous masses; secondly, in usually affecting one lung only, or even one lobe; while phthisis, properly so called, can only be confined to one lung in its earlier stages. Some specimens of fibroid and indurated lung, with cavities much like what have just been described, are found on examination to contain tubercles.

FIG. 109.



Fibroid or adenoid tissue from an indurated lung, showing abundant lymphoid corpuscles and fibrous intercellular substance. It much resembles the tissue produced in chronic inflammation of a lymphatic gland. (See pp. 154 and 418.)

Dr. Williams describes a chronic form of pneumonia, in which the hepatized portion, owing to the thickening of individual vesicles, assumes an oolitic aspect. He is of opinion that consumption may originate in this species of pneumonia without the pre-existence of any distinct tuberculous disease. It is not impossible that some of the fibroid contractions of the pulmonary tissue, which we meet with, particularly at the apices of the lungs, may be due to an arrest of chronic pneumonia as well as to previous pleuritic inflammation; we occasionally meet with depressions in otherwise healthy lungs, which are unconnected with emphysema, and for which no other explanation can be offered than a foregone inflammatory condition of the interstitial tissues of an aplastic character, an opinion in which we are also borne out by the authority of Dr. Williams.*

COLLIERS' PHTHISIS, ANTHRAKOSIS, PIGMENTATION OF THE LUNG.

Closely connected with ordinary chronic pneumonia are those destructive diseases of the lung which are caused by the inhalation of irritating substances in the form of dust. The best known disease of this kind is that described as the "Colliers' Phthisis," or, as it has been called, anthrakosis. The lungs of colliers are often found after death to be exceedingly full of black pigment, and there is no doubt that this is carbonaceous dust inhaled during life. Objections were at one time made to this conclusion by some German pathologists, but the discovery by Traube in a lung of this kind of a fragment of undoubted black coniferous wood, which must have come from coal, showed the correctness of the opinion always entertained in this country. Beside blackness, the collier's lung shows induration, condensation, and often the formation of cavities. Changes very similar to these are produced in the lungs by the inhalation of various kinds of dust, as oxide of iron, the dust of copper mines, that caused by grinding, stone working, flax dressing, &c., though in these the black colouring is less marked. The characters common to all these cases are summarized by Dr. Greenhow* as consisting in thickening of the pleura, increase in the density of the lungs, and formation of patches of consolidation of various sizes in their substance. These nodules vary from a pea to a walnut in size, and are either iron grey or nearly black in colour: all trace of the vesicular structure of the lung is lost in them, but the smaller are formed of the thickened walls of bronchioles. Even the less altered parts are traversed by bands of connective tissue. The lungs, when cut into, exude more or less of a black fluid, resembling Indian ink, which is most abundant in the lungs of miners. The bronchial glands are also enlarged and black, and yield a similar black fluid. Minute examination shows two principal changes in such lungs, viz., thickening of the alveolar septa, causing in the more altered portions almost complete obliteration of the alveoli; and, secondly, deposition of pigment in small granules or irregular masses through the interstitial connective tissue; *i.e.*, in the walls of the air cells, and in linear tracts corresponding to the interlobular septa, to the course of vessels and air tubes, and to the subserous connective tissue.† Black pigment is also found in cells, both in mucous corpuscles and in ciliated epithelium. In the cases described by Zenker, where persons were in the habit of inhaling dust of oxide of iron, *red* pigment was found in corresponding situations.

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* "Principles of Medicine," p. 313. London, 1843.

Chemical analysis shows that the black lungs contain a quantity of black matter insoluble in any acids, *i.e.*, carbon: the lungs of miners contain notable quantities of silica. The disease is then evidently to be described as a chronic destructive pneumonia produced by irritating particles. The presence of excess of carbon in the lungs of stonemasons is, notwithstanding Dr. Greenhow's explanations, not quite accounted for; but the black pigment found in the lungs of all persons after death is clearly shown to be derived from the smoke of fire, candles and lamps, which everyone inhales more or less. It is an old observation that this pigment is absent in the lungs of children, and most abundant in those of old people.

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* "Trans. Path. Soc.," vol. xx. p. 51.

† We may just point out that these are the situations of the lymphatic paths, by which fine particles would naturally find their way through and out of the organ.

ADVENTITIOUS PRODUCTS IN THE LUNGS.

TUBERCLE.

We have already given (p. 193) a definition of a tubercle such as it is found in various organs and parts of the body. Tubercles thus defined occur in the lungs in various pathological conditions, but the term has been applied with greater laxity than in the case of most other organs, and many objects met with in the lungs, to which this name has been given, do not conform to the definition of a tubercle given above; that is, are not like what are called tubercles in other parts of the body. Speaking generally, the name has been applied to small (that is, not much more than one-eighth of an inch in diameter) solid bodies, harder and more resistent than the ordinary lung tissue, grey or yellowish in colour. To such bodies the term granule or granulation has also been applied, but since a very special meaning has been attached to this term also, the word nodule appears to be the only one that can be used without involving any theoretical explanation.

The nodules, which have been called tubercles in the lung, are of the following classes:—

1. *Grey Miliary Tubercles.*—These are tubercles such as they occur (for instance) on the serous membranes, or true tubercles as formerly defined. They may occur simply, or else complicated and enlarged by various changes in the surrounding lung tissue. When uncomplicated, they most commonly occur in large numbers spread through the whole or great part of one or both lungs; and the most typical examples are found in the disease called "acute miliary tuberculosis," when they are present in other organs also. These are the "grey granulations" of Bayle and Louis, and only partly identical with the grey tubercles of Laennec, which have been often called grey miliary tubercles.

2. *Peri-bronchial Granulations.*—These are small, hard masses formed round the smaller bronchi, first clearly distinguished by Virchow from miliary tubercle, though described by Carswell.

3. *Broncho-Pneumonic Granulations.*—The objects which Laennec for the most part described under the name of grey tubercle, or grey granulation, though he also included under this term the true miliary tubercle, and the following class. They are the product of catarrhal broncho-pneumonia, to be described hereafter.

The above are the chief classes of objects known as tubercles, but the following must also be mentioned as likely to be mistaken for them:—

4. *Small Syphilitic Nodules.*—The characters and history of these

are not as yet very well known, but they appear to resemble tubercles in many respects.

5. *Small Fibroid Granulations* of uncertain origin are sometimes found scattered through the lung where the history and absence of accompanying changes forbid the supposition that they are tubercles. Such granulations are far more common on the pleura and other serous membranes, and those occurring in such situations may be taken as a standard of comparison for the ill-defined nodules in the lung.

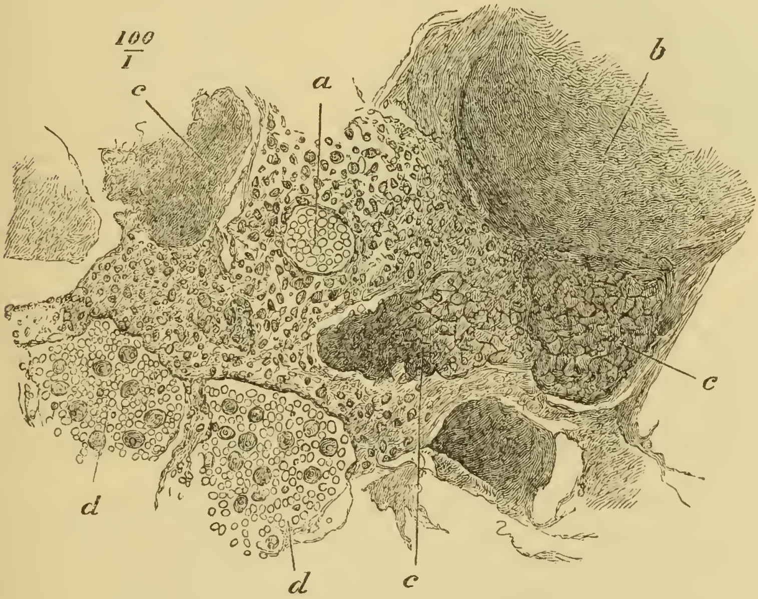
Grey Miliary Tubercles.—These when met with in an uncomplicated form (as in acute miliary tuberculosis) are hard, resistent, translucent bodies, projecting on the cut surface of the lung, of a pale grey colour, sometimes yellower and more opaque in the centre. Their shape appears at first sight round, but a low magnifying power, or even close examination with the unaided eye, will suffice to show that they are really angular, and that the angles are prolonged into the intervesicular or interstitial structure of the lung. The average size may be estimated as about that of a pin's head, the extremes varying from the limit of vision to the size of a hemp seed, or $\frac{1}{8}$-inch in diameter. If larger than (or perhaps even as large as) this, it is extremely probable that the apparent size of the tubercle is increased by inflammatory or hypertrophic change in the surrounding parts, which we may not be able to detect with the unaided eye.

The microscopical examination of these tubercles, especially in sections of the lung containing them, shows that they consist of small cells, sometimes containing nuclei, often not (and in this case undistinguishable from nuclei), generally arranged somewhat concentrically round one or more centres. The more external parts usually consist of larger cells, the central parts of smaller, or the latter may even be degenerated and amorphous. Sometimes by tearing the structure out with needles larger many-nucleated cells, like "myeloid" cells, may be demonstrated. Intercellular substance is less easy to make out than in tubercles from some other parts.

Complicated Miliary Tubercle.—The tubercle may, as we have said, give rise to inflammatory or hypertrophic changes in the surrounding tissue, and the nature of these will depend upon the disposition of the organ in which the tubercle occurs. In the lung the changes thus produced are chiefly of two kinds—(1) catarrhal inflammation, (2) hypertrophy of connective tissue, chiefly resulting, not in the formation of vascular fibrous tissue, but of cytogenous or adenoid tissue, which gives the appearance known as fibroid degeneration, or induration. Hæmorrhage also frequently occurs, but its precise relation to the production of tubercle is not known; and breaking down, or the formation of cavities, which will be further discussed. In many cases where all or any of these changes accompany tubercle, it is impossible to say whether they have preceded or followed it.

The most important histological fact is that the tubercle is not usually found within the air cells, but extends along their walls and partitions; and though it may occupy the space of several air cells, these are flattened or compressed, but not filled up with the new growth, or their contents may be merely converted into an amorphous mass. The tubercle is therefore interstitial, but we regard it as till now quite undetermined whether it is formed in the walls of minute arteries (as in the pia mater), or veins (as in Fig. 110), or in the walls of minute bronchi, or in the cavity of lymphatic vessels (Rindfleisch). Another important fact is that the tubercle, when large enough to be visible to the naked eye, contains no vessels. In an injected specimen (Fig. 45, p. 194), it may be seen that these become obliterated on reaching the tubercle.

Fig. 110.

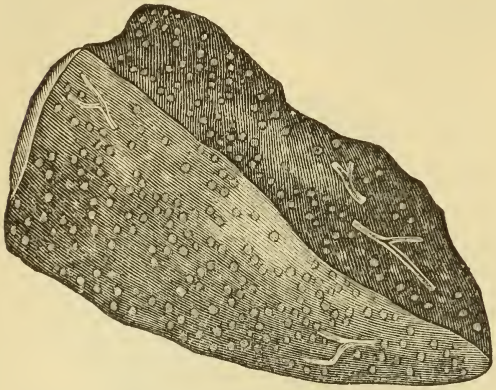


Section of lung, showing miliary tubercle complicated with inflammatory changes in surrounding parts.

a. Vein surrounded by the nucleated tubercular growth, which extends along the alveolar septa. *b.* Bronchus filled with degenerated (caseous) inflammatory products. *c.* Alveoli containing fibrin and other products of inflammation, partly degenerated. *d.* Alveoli containing blood from recent hæmorrhage, and catarrhal cells. (From an original preparation.)

Conditions of Lung in which Miliary Tubercle occurs.—This form of tubercle may occur generally scattered through the lung or confined to one spot. When it is generally distributed, we may conclude that all the tubercles were formed at once, or during a short space of time, as the consequence of an acute affection, and tubercles of the same age are generally found either on the pleura or in some other organs, as the pia mater, kidneys, peritoneum, &c.—that is to say, there is acute general tuberculosis. In such cases (as was said before) we can generally find some trace of previous disease, chiefly of the type of degenerative inflammation, resulting in the formation of caseous matter. But sometimes a simple inflammation seems to be the precursor of tuberculosis, and sometimes no antecedent disease is discoverable. In the lung, however, miliary tubercle, though generally distributed, may occur as the sequel of disease in the same organ; a cavity or mass of degenerated matter being found at one apex (for instance), while one or both lungs are studded with miliary tubercles. The intervening tissue is often quite unaltered, sometimes extremely hyperæmic, though crepitant and otherwise natural, and, less frequently, actually pneumonic. This mode of occurrence of miliary tubercle may therefore be an accompaniment of phthisis, but is not necessarily so.

Fig. 111.



Miliary tubercle, scattered throughout the pulmonary tissue, forming translucent, greyish, and circular points of the size of pins' heads.

On the other hand, the *local* occurrence of miliary tubercle in the lung is always in combination with some form of phthisis. We may find scattered tubercles surrounding a large cavity, or imbedded in masses of grey fibroid tissue, or mingled in various proportions with destructive or indurative change, as will be shown in speaking of the different forms of phthisis.

PERI-BRONCHIAL GRANULATIONS.

These bodies have very often been mistaken for tubercles in a certain stage of retrogressive change. They are hard dark grey nodules, with a yellowish centre, the latter having often been regarded as a degenerated portion of the tubercle. Close examination, however, shows that this yellow centre is really a perforation, and represents the cross section of a small bronchial tube, the thickened walls of which cut across, with some surrounding consolidated tissue, constitute the nodule. The hollow of the tube contains either ordinary inflammatory products, mucus, pus, &c., or more generally the same products which have already undergone degenerative decay, or *caseation*. These products may completely fill up the tube. More information is in some cases obtained respecting the structure of these objects by cutting open the bronchial tubes, beginning with the larger. It will then be found that there is, even in the larger tubes, bronchitis, and that this most probably appears more intense on reaching the smaller tubes, while in some cases the degenerated secretion and inflammatory products appear on the inner surface as what was formerly called "yellow tubercle," or earlier still "scrofulous matter." This was admirably represented by Carswell in 1838,* though he was evidently wrong in regarding this process as the characteristic one in tubercular disease, or in all forms of phthisis.

The thickening and induration of the bronchial wall are chiefly owing to an infiltration with small cells, which are very prone to undergo decay and become granular, so that their structure becomes undistinguishable. In early stages, however, we believe that this cellular structure is really tubercle, with some accompanying hypertrophy, and that the peri-bronchitic granulation may be described as *peri-bronchial tubercle.* The apparent size of the nodule, as seen with the naked eye, is also partly made up of inflamed and consolidated lung tissue.

The only account which can be given of the origin of peri-bronchial tubercle is, that the inflammation affecting the mucous surface of the bronchi, which may at first be ordinary bronchitis, extends to the submucous tissue and the bronchial walls. The inflammation excited in these parts is not suppurative, but chronic and indurative, being moreover, as we believe, always accompanied by the production of tubercles. The whole process may be called *chronic peri-bronchitis*, or more specifically, *tubercular peri-bronchitis.*†

Peri-bronchitis is always accompanied by broncho-pneumonia, inflammation spreading to the surrounding air cells, and also to those which are in communication with the bronchial tubes affected. It is also very common to find grey induration (production of cytogenous or fibroid tissue) of the surrounding parts, and in general those forms of phthisis in which induration preponderates, are those in which peri-bronchitis chiefly occurs. As might be expected, true miliary tubercles are also frequently associated with the peri-bronchial granulations. Peri-bronchitis unaccompanied by tubercle is, we believe, very rare in the human lung, if, indeed, it ever occurs; but according to Virchow it is a common disease in dogs.

BRONCHO-PNEUMONIC GRANULATIONS.

The origin of these nodules is from the catarrhal broncho-pneumonia, of which we have already spoken. We only wish here to recapitulate certain characters belonging to them, some of which have led to their being confounded with tubercles, and others by which they may be distinguished from them. They resemble tubercles in being hard, resistent, grey, and often translucent, though readily becoming yellow and opaque.

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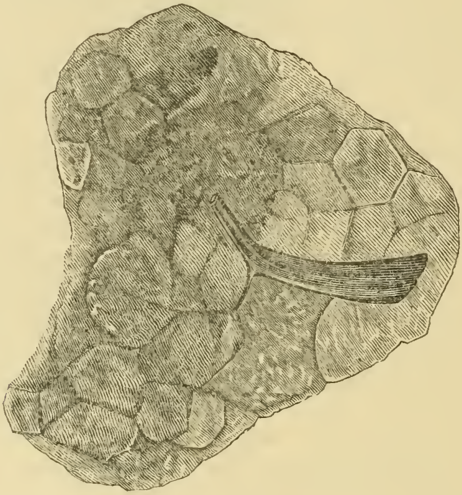
* "Illustrations of the Elementary Forms of Disease."

† This epithet is authorized even by Virchow, who has so clearly distinguished these structures from miliary tubercle.—"Die Krankhaften Geschwülste," vol. ii. p. 648.

The points of difference between these structures and tubercles are as follows:—They are somewhat larger, being generally as much as one-eighth of an inch in diameter, and often more, so that their minimum size is equal to, or greater than, the maximum of miliary tubercles. Then the arrangement of broncho-pneumonic masses is quite characteristic, being (as we have already pointed out) determined by the fact that the air spaces connected with one terminal bronchiole are simultaneously affected, and hence the group is like a cluster of nodules attached to a single stem. When such a group of granulations becomes confluent, it forms a mass which may be grey, but is more likely to have become yellow and opaque, while it has the characteristic shape of lobular pneumonia. On the pleural surface these granulations (called racemose by some writers) have a polygonal outline (Fig. 112). True miliary tubercles never become confluent into a considerable mass of the same kind as themselves, though they may be united by inflammatory or fibroid products. Hence nodules which are in immediate connection with masses of the same structure must be broncho-pneumonic, not tubercular. Again, the parts surrounding the broncho-pneumonic granulation may be, if not actually consolidated, in the condition described by Laennec as "gelatinous infiltration," that is, condensed and imbued with serum in a condition like an exaggerated œdema. Also, for similar reasons to those just given, these granulations are seldom found scattered singly through a comparatively healthy parenchyma, as we often find miliary

Fig. 112.



Hexagonal appearance caused by the mutual pressure of the air cells, filled with yellow cheesy matter, with obliteration of the bronchioles leading to the lobules. Magnified 60 di.

tubercles. As to their consistency, also, broncho-pneumonic nodules are, though hard, less hard than miliary tubercles, and have not quite the same almost cartilaginous appearance. When found scattered through healthy lung tissue, they constitute the disease called miliary hepatization by Virchow, which is one of the forms of so-called acute tuberculosis, seen in children.

Microscopical investigation, of course, reveals the fundamental distinction; the broncho-pneumonic nodules giving, on scraping, large catarrhal cells, some in a state of fatty degeneration; glomeruli, granular matter, &c., while from the miliary tubercle we obtain, by the same, only a few of the cell forms already described. While on section it will be seen that in the one case the solidification is caused by accumulation within the air cells; in the other by an interstitial growth.*

Combination with Tubercles.—As might be expected, broncho-pneumonic granulations and tubercles are very frequently combined. Sometimes the two can be clearly distinguished; sometimes, on the other hand, the tubercles may be, so to speak, concealed in larger broncho-pneumonic masses. Rindfleisch states that they may be found probably in all cases where broncho-pneumonic granulations exist, though perhaps not in large numbers.†

We need not now dwell upon the other objects mentioned above as likely to be confounded with miliary tubercles, but proceed to arrange the observations already made under the different forms of phthisis.

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* This distinction has for the most part been maintained in the controversies of the last few years on the subject of tubercle. This has been generally regarded as something interstitial, and thus distinguished from pneumonic or catarrhal products; though the term might sometimes be applied to diffused growths as well as to discrete granulations. Even Dr. Wilson Fox, who in the recent discussion at the Pathological Society ("Path. Trans.," vol. xxiv.) expressed views widely different from those here adopted, "agrees with Virchow that tubercle is not an infiltrated product," and refuses this name to pneumonic products occupying the interior of the alveoli (*op. cit.*, p. 364). It was agreed by most speakers in the discussion which followed, that two chief classes of elements were found in pulmonary phthisis, a small-celled growth in the walls, and a large-celled growth in the cavity of the alveoli—the former alone, if any, being tubercle; though some speakers proposed to call both inflammatory, and reject the name tubercle altogether. While these sheets are passing the press, the sharpness of this distinction has been destroyed by researches which tend to restore the name of tubercle to infiltrated or intra-alveolar products. Dr. Klein, in a communication to the Royal Society on the lymphatics of the lung, describes the appearances in several cases of so-called acute tuberculosis of the lungs, where he found tubercle beginning with the characteristic myeloid cells, within the alveoli, on their free surfaces. These cases were no doubt such as are referred to in the text under the name of miliary hepatization. The condition is what was meant by the term infiltration used by Laennec, and described by the early microscopical observers. If it should be found to obtain generally, doubtless the definitions given above would require modification, and much of what we have called broncho-pneumonia might be called tubercle. We should then have to fall back upon the distinction insisted upon in the next chapter between such forms of (tubercular or phthisical) disease as are distributed by the respiratory channels, and show a corresponding anatomical arrangement; and those which are propagated by the vascular systems, sanguineous or lymphatic, agreeing in their distribution with these. This distinction is confirmed by Dr. Williams's explanation (arrived at on very different grounds) of the two forms of tubercle or phthisis, "as (1) lymphatic, which is miliary, infective or scattered; (2) inflammatory, which is different in form, and local in extent." ("Path. Trans.," vol. xxiv. p. 340.)

† "Pathologische Gewebelehre," p. 349, 1st ed.

CHAPTER XXXIII.

PULMONARY PHTHISIS AND NEW GROWTHS.

By phthisis we understand a chronic disease leading to partial destruction of one or both lungs. This change never occurs without inflammation; seldom without the presence of tubercles, and is frequently attended by the production of fibroid tissue.

The controversies about pulmonary phthisis have of late years in great part turned upon the question whether there are several forms, or whether all are reducible to one;—the question, as it is called, of the unity or multiplicity of phthisis. According to the one school (which agrees with the doctrines of Laennec), all changes in a phthisical lung are due to the deposition or production and subsequent changes of one particular kind of material, viz., tubercle. Another school regards some cases of phthisis as wholly due to inflammation, and other cases as partly inflammatory, partly due to the changes of tubercle. Again, of the non-tubercular changes, two types are distinguished, namely, caseous (catarrhal) and fibroid.

We believe that the varieties of phthisis depend partly upon the presence or absence of tubercles and the predominance of the changes due to these over inflammatory and degenerative changes, or *vice versâ*, but only partly; and that another important cause of the different appearances is the mode in which the disease becomes disseminated through the lungs. When it is associated from the first with inflammation of the bronchi and subsequent changes in the alveoli, it has the appearance characteristic of disease distributed by these channels, viz., that of broncho-pneumonia or lobular pneumonia; which again is also the appearance characteristic of a partially inflated fœtal lung, an appearance produced by the fact that the affected portions are such as communicate with particular air-passages. This will be the case whatever be the nature of the material filling the alveoli; whether it be a purely inflammatory product or something specific, called tubercle. This constitutes the catarrhal form of phthisis. In other cases it does not appear that the disease has followed in its distribution the channels of respiration, but rather those of circulation, sanguineous or lymphatic. In such cases the "tubercles" will appear scattered through the lung without any reference to the bronchi or the anatomical divisions of the lung dependent upon them. Their distribution will then appear quite irregular, though minute examination may show that a certain number of the granulations are formed around blood-vessels in other perivascular lymphatic channels; while there may be other evidence of the participation of the lymphatics, such as the presence of tubercles in the pleuræ, which are known to contain very numerous lymphatic channels leading from the lung. This constitutes the infective form of phthisis, and agrees in its mode of distribution with acute miliary tuberculosis formerly spoken of, being, in fact, essentially the same disease become chronic. These two extreme types of disease are sufficiently contrasted when seen absolutely unmixed, but this very rarely happens; almost all cases presenting some features of both morbid processes. The catarrhal form becomes in fact complicated with tubercles, produced, as is thought, by local infection; and the tubercle in the infective form never fails to produce secondary inflammation round it. Hence, in a chronic disease, whichever may have been its mode of origin, the appearances will be ultimately the same, and the catarrhal and infective phthisis will then be not only indistinguishable but identical. We find in both small-celled growth (what is usually meant by tubercle) in the walls of the alveoli and interstitial tissue; while in the alveoli and bronchial tubes are seen masses of larger cells, fibrin, and other so-called inflammatory products; nor is there anything to tell us which of these represents the original disease.

In all chronic forms of phthisis there is some fibrous induration or production of fibroid tissue, but this is sometimes the preponderating, or even the exclusive change, constituting the disease called fibroid phthisis.

FORMS OF PHTHISIS.

We recognize the following forms of the disease commonly described as phthisis of the lung:—

1. *Catarrhal Phthisis or Caseous Pneumonia.*
2. *Mixed Phthisis.*
3. *Tubercular Phthisis.*
4. *Fibroid Phthisis.*

The form of disease in which grey miliary tubercles are found scattered through comparatively unaltered lung-tissue is sometimes represented as a form of phthisis, but since there is no actual destruction of the lung, it does not come under our definition of this disease. We have already described it as acute general tuberculosis affecting the lung, and have nothing to add to that description.

The differences between these forms depend essentially upon the mingling, in different proportions, of tubercles properly so called, with the products of acute or chronic inflammation. The first form of phthisis is essentially a pneumonia, with peculiar history and tendencies; the second is similar pneumonia with miliary tubercles superadded; in the third the disease appears to begin with the formation of tubercles, but soon to become complicated with pneumonic processes. The second and third forms will then, when they have reached a certain stage, be practically identical, and their distinction will be a matter of inference, not of observation. They will therefore be described together. The production of fibroid tissue, or induration from chronic inflammation, may be a part of either of these diseases; but in the form called fibroid phthisis it is the predominating change.

I.—CATARRHAL OR BRONCHO-PNEUMONIC PHTHISIS; CASEOUS PNEUMONIA; SCROFULOUS PNEUMONIA (VIRCHOW).

This form of disease, though it undoubtedly exists, we believe to be, in an unmixed form, rare. When it occurs it is as a sub-acute disease, sometimes following an acute febrile complaint, as measles or simply acute bronchitis. When chronic we believe it to become almost invariably complicated with the production of tubercles, and usually of fibroid tissue also; the disease then becomes mixed phthisis. At the same time there are cases of chronic *limited* broncho-pneumonia when the presence of tubercle cannot always be traced; and we thus have two actually occurring forms of the disease—(1) acute and general; (2) chronic and partial. In unmixed catarrhal phthisis, we have a considerable portion of one or both lungs, sometimes nearly the whole, consolidated; while other portions are softened or actually converted into cavities; and there is a certain proportion of normal lung tissue. The consolidated portions are to a large extent yellow, dry and friable, in fact caseous. This condition is the yellow tubercular infiltration of Laennec, the scrofulous pneumonia, caseous pneumonia, or tubercular pneumonia of different modern writers. Other portions will be in the earlier stage of this affection, that is to say, a grey or pinkish grey hepatization, with the characters of simple catarrhal pneumonia, said to be the "grey tubercular infiltration" of Laennec; and again there will be firm œdema or gelatinous infiltration.

The three conditions just mentioned are the three stages of degenerative or caseous pneumonia; the same affection which, here found alone, accompanies the production of tubercles in chronic phthisis. The yellow colour of the last stage is due to fatty degeneration of the catarrhal products contained in the alveoli. This disease is usually lobular, or develops from broncho-pneumonia, but is said to develop from lobar pneumonia also. The diseased portions are sometimes separated from healthy lung tissue by a tolerably definite line. (Fig. 113.) At other times small masses of granulations show the gradual extension of the morbid process. The formation of cavities seems to take place by necrosis and softening of masses of caseous material; attributed by some to the mutual pressure exercised by the masses of cells and inflammatory products which choke the alveoli, by others to obstruction of vessels. Sometimes, though not generally, active suppuration takes place through the lung, and numerous small cavities are formed by a sort of purulent infiltration.

Ætiology.—This affection, though unaccompanied by miliary tubercles is evidently closely related to tubercular disease. This is sometimes shown by one lung being in the condition of caseous pneumonia, while the other contains tubercles; or as we have seen in two or three cases, by the presence of tubercles in the pleura covering a lung affected with this disease. It has been supposed that even when no tubercles are discoverable they must nevertheless have been present,* but we do not see sufficient reason for this supposition; at the same time it is very probable that the two affections may be due to the same cause, and may be thus spoken of as part of the same disease; so that if any morbid "poison" should ever be shown to be the cause of tubercle, that will probably be found to produce, in some cases, "caseous pneumonia" also. The cases we have seen have been chiefly sequelæ of measles or acute bronchitis. Others are given by MM. Hérard and Cornil (*op. cit.*, pp. 498, 504).

This is the disease which is a consequence of, or perhaps constitutes, one form of catarrhal broncho-pneumonia. Why in one case that affection should pass away without leaving any trace behind; and why in other cases it destroys the lung, we do not know. Two suppositions may, however, be made. First, that the constitution of the patient, that is some structural peculiarity in his tissues, is the cause of the difference; or, secondly, that there is some specific poison or morbid material contained in the blood, which causes the bronchitis and pneumonia which constitute phthisis to differ from simple inflammations of the same kind.†

CHRONIC LIMITED BRONCHO-PNEUMONIC PHTHISIS.

In this which is the commonest form of phthisis, at least among the non-fatal cases, the disease begins in the apex of the lung; is often confined to that part, producing the well known signs in the infraclavicular region known as those of "early phthisis." If we have an opportunity of examining the disease in an early stage we find a soft yellowish mass of degenerated matter probably surrounded by or continuous with a very few pearly granulations of broncho-pneumonic character such as have been just described. If the disease have lasted longer there may be some grey induration, and dilated bronchial tubes, the latter containing, perhaps, some putty-like matter. If the disease be distinctly obsolete or obsolescent there will be also puckering of the pleural surface; and probably nothing else but fibroid tissue, or, perhaps, a little putty-like matter contained in a bronchial cavity. In these latter stages nothing that can be defined as a tubercle is to be found; nor is there any evidence that tubercles have been there and been absorbed. In earlier stages (which only chance opportunities can reveal to us) tubercles are at least in some cases absent. Hence the probable (though not absolutely certain) conclusion is that all the appearances just described may be produced entirely without the intervention of tubercle. Formerly it was thought that all the changes just described came from a primary deposit of tubercle; and though we cannot absolutely disprove this assertion in all cases, we can say that it is not a positive conclusion, and not supported by such evidence as an inference of that nature demands. As, however, the production of tubercle is always likely to occur, this form of disease passes insensibly into mixed phthisis.

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* Hérard and Cornil, "La Phthisie Pulmonaire," pp. 144 and 492. Paris, 1867.

† Whether the catarrhal pneumonia, which is in most cases the cause of phthisis, is a different disease from ordinary catarrhal pneumonia; whether it is not in many cases so to speak "specific," constituted, perhaps, by a certain "imperfection" of the textural elements of the lung substance; this may at least for the present be regarded as an open question. (Oppolzer, "Vorlesungen über specielle Pathologie und Therapie," vol. i. p. 645.)

II. AND III.—MIXED PHTHISIS AND TUBERCULAR PHTHISIS.

The first of these forms, which includes by far the larger proportion of all cases of phthisis, was formerly regarded as entirely due to the formation and subsequent changes of tubercles; lately, especially by German writers, it has been represented as purely inflammatory in its origin, an opinion long ago defended by the late Dr. Addison.*

If we regard tubercles as signifying something beyond and different from inflammation, then this way of looking at the question is certainly too narrow, since tubercles may almost always be found in chronic cases.

On the other hand, when tubercles are once formed, they always become complicated with inflammatory changes. The phenomena will then be the same as if the disease had begun with inflammation and tubercles had been superadded. For these reasons they find it impossible to distinguish between the third and fourth forms when the disease has become chronic, and we feel compelled to describe together the mixed phthisis which is in its commencement inflammatory, and tubercular phthisis, which becomes inflammatory, as *Chronic Phthisis.*

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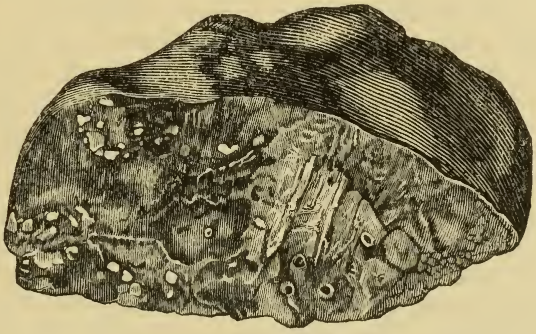
* See also Wilks's "Lectures on Pathological Anatomy," 1859, p. 240.

CHRONIC PHTHISIS (MIXED AND TUBERCULAR).

Chronic phthisis nearly always begins at the apex of the lung, the exceptions to this rule being found chiefly in children. If we happen to light upon a lung in an early stage of the disease we find a larger or smaller portion of the apex consolidated, the solid tissue usually yellow, dry, and somewhat caseous, like the whole lung or larger masses in the catarrhal phthisis referred to just now.

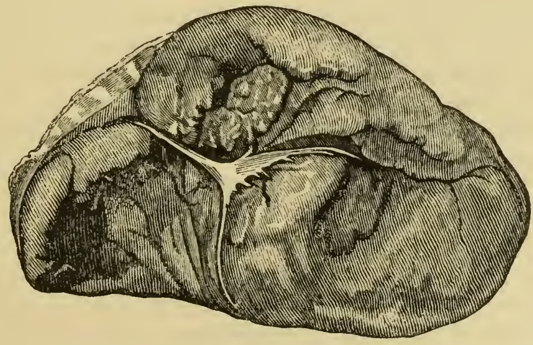
FIG. 113.



The apex of a lung affected with caseous pneumonia; the limitation of the disease was defined by a sharp line.

The affected portion is sometimes separated by a sharp line from the healthy lung, sometimes a border of broncho-pneumonic granulations is seen. There is not necessarily any miliary tubercle.

FIG. 114.



Cicatrix at the apex of a lung, resulting from the previous arrest of tubercular disease.

So slow is the progress of disease that further changes take place before any large portion of the lung is affected. The centre of the consolidated portion becomes softer and more cheesy, while the outer portion becomes harder and more fibroid. In this way begin the two most important processes of chronic phthisis, viz., softening or breaking down and induration. The former process may go on to the formation of a cavity, as will be shown hereafter. If the latter preponderate, the degenerated tissue may become surrounded by a firm capsule and thus become obsolete (Fig. 114), and the disease may thus be arrested, as is more minutely described below.

If the disease extend over the whole of a lobe a much greater variety is seen in the products to which it gives rise. Besides some masses of caseous matter, we shall find fibroid tissue, peribronchial thickenings, and broncho-pneumonic granulations, besides, in all probability, true miliary tubercles. If these are very numerous the appearances presented will not be distinguishable from those seen in cases in which the disease commences with the production of miliary tubercles or chronic tuberculosis. It is impossible to give any description of the manifold appearances which result from the intermixture of all these products, and we can only describe separately the chief morbid processes on which they depend.

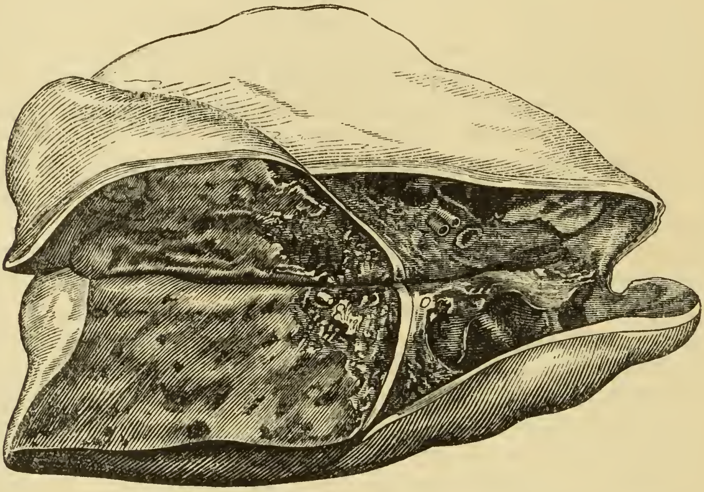
Necrosis, Caseation, and Breaking-Down.—The death and degeneration which lead to the formation of caseous masses affect both the new formations, whether inflammatory or tubercular, and the original tissue. Various explanations have been given of this decay, or "necrobiosis," in different cases. As to the inflammatory products which choke up the alveoli, their history is, up to a certain point, the same as that of ordinary pneumonic products, and, like these, they might undergo fatty degeneration, softening, and elimination. They do, however, become dry, cheesy, and crumbling. This change has been explained by the mutual pressure exercised by the newly-formed cells tightly packed together in the air spaces, which interferes with their nutrition. If this accumulation of inflammatory products continue so that the pressure is still further increased, the blood-vessels become obliterated, and more rapid necrosis, leading to softening, occurs, which involves also the lung tissues. According to Virchow, the early decay of phthisical products is a special property of the elements formed in all the diseases called "scrofulous." According to others, it is the property of the newly-formed elements in so far as there is a general diathesis or constitution of the body which causes all new formations to be incapable of higher development and liable to early degeneration and death. In all these theories something more is wanting to account for the actual death of the tissues themselves. This is explained by an old observation, lately revived by Dr. Wilson Fox, that the vessels, both capillaries and smaller arteries, in parts affected with tubercle, are often found obliterated. Hence the obliteration and destruction of vessels is regarded as the cause of caseation and necrosis, both of the morbid products and of the tissues of the part, this obliteration being caused by growth of tubercle in the vascular wall, and being, as we have shown before, an essential feature in the growth of tubercle (p. 194).

Another source of caseous material must not be overlooked, viz., the degeneration of effused blood. This gives rise, as is well known, to dry crumbling material, which may soften and liquefy in the same way as the cheesy masses of phthisis, and as a matter of fact capillary hæmorrhage into the alveoli often takes place, as seen in Fig. 110, so that in this way a certain amount of caseous matter may be produced. Indeed, an old hæmorrhagic infarction (p. 474) has, at a certain stage, much resemblance to "crude tubercle."

Formation of Cavities or Vomicæ.—Destruction of the lung with formation of cavities is the most constant and important of morbid changes in chronic phthisis. It results partly from the necrosis or slow decay just described, and partly from inflammation and suppuration. So the phthisical cavity or vomica is not precisely an abscess, nor merely the result of local death or necrosis. It should be remembered that the lung tissue is exposed to the air and to the putrefactive influences brought by it, so that decay will be more rapid and more resemble ordinary putrefaction (as in fœtor) than necrosis in secluded parts of the body. In vomicæ there have often been found the minute organisms (micrococci and bacteria) which accompany putrefaction.

After the first production of tubercle is completed, the morbid diathesis may, as it were, have exhausted itself and the process enters into a state of abeyance, all active symptoms being arrested; or the inflammatory action is perpetuated and the course is continuous to the fatal termination. In the former case every recurrence of bronchitic or pneumonic inflammation finds a nidus of tubercle around which fresh matter is deposited, or which becomes an additional source of irritation. In both cases a process of fusion takes place, serum and pus are secreted from the parts in which the morbid deposit has been effected, the intervesicular septa are more and more absorbed and broken down, the softened matter finds its way into the larger bronchi and is expectorated, a mixture of pus, mucus, melanotic, oily and granular matter, interspersed with epithelium and the elastic fibres of the lungs, and the result is a cavity in the pulmonary tissue. The walls of this cavity may be more or less ragged, and be more or less lined with caseous matter, or present no traces of it, according to the date of its formation. Tubercular cavities or vomicæ were formerly often mistaken for genuine abscesses, and were sometimes called ulcers. We find them in all numbers, there may be but one, or so many as to give the entire lung a riddled or honeycomb appearance when cut into; it is rare, as Laennec observes, to find a single cavity. They vary equally in size, from a pea to a man's fist, and more. The communication between the abscess and the bronchus passing out of it resembles a fistulous opening, the peculiar relation of which to respiration causes many of the phenomena of auscultation. The excavation is commonly surrounded by crude and miliary tubercles which gradually soften, are then discharged into the main cavity, giving rise to the anfractuosities which we commonly observe. The excavations are often traversed by bands of pulmonary tissue infiltrated with tubercular matter, and compared by Laennec to the columnæ carneæ of the heart. These bands often contain branches of the pulmonary artery, the walls of which are always thickened and infiltrated with tubercular products, and the channels of which are usually obliterated. Less frequently the vessels remain pervious and become enlarged into aneurismal dilatations, from which fatal hæmorrhage sometimes occurs. The bronchules, and successively the larger air-tubes, are subjected to the same destructive agency, until the power of resistance caused by the stronger walls of the bronchi of the first and second order is too great to be overcome by the morbid process.

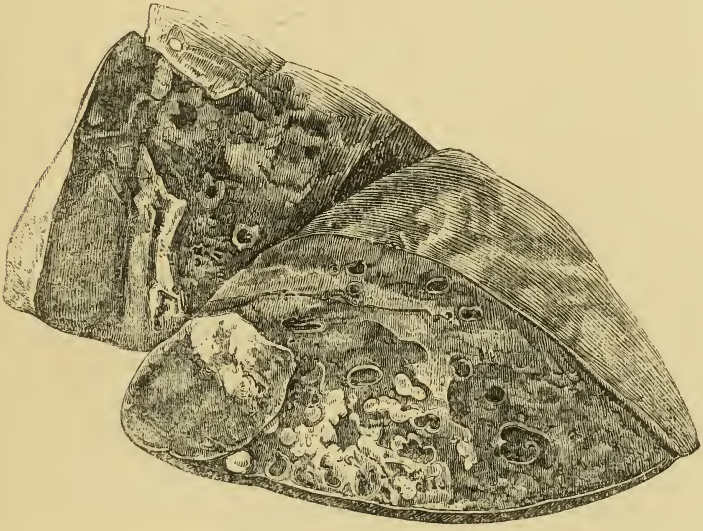
FIG. 115.



A lung, exhibiting extensive tubercular disorganization throughout its upper lobe, which is almost converted into one ragged cavity. The pleura is very much thickened; intimate adhesion has taken place between the upper and lower lobe, and the tubercular disease is seen encroaching upon the latter.

Instead of a mere uneven rugged surface, as if the lung had been mouse-eaten, the walls of old cavities often present a uniform velvety appearance, and are invested by a false membrane, which may assume a considerable thickness, but at the earlier periods of disease is of slight consistence and easily separable. The tissue beyond the lining membrane may be in a healthy condition, or present tubercular and inflammatory products, but generally contains much melanotic carbonaceous matter. This false membrane resembles what has been called the pyogenic membrane of an ordinary abscess. It may produce on the one hand pus, on the other hand permanent connective tissue. The former is shown by the profuse expectoration so often seen in phthisis, the latter in the firm walls which enclose old vomicæ in the lung and the induration of surrounding tissue. The greater smoothness of the internal surface, and the firmness of the walls, distinguish old from recent cavities. Dilatations of the bronchial tubes complicate and enlarge cavities in varying degrees. Many cavities doubtless commence in bronchial dilatations, accompanied or not by ulceration of the walls.

FIG. 116.



The apex of a lung containing numerous cavities, with tubercular and caseous products intervening. The large cavity, and several of the smaller ones, are lined with an adventitious membrane.

In the majority of instances both lungs are found to present excavations. Louis states that in one-sixth only of his total cases of phthisis, they were limited to one or the other lung, and when present on both sides were of different size; in somewhat less than one-tenth of his cases, both lungs were the seat of enormous excavations, equally large on both sides, and in another tenth, where the cavities presented but small or moderate dimensions, these dimensions were the same in both organs. Large cavities (of the size of a goose's egg or a clenched fist) Louis found to occur in about one-half of the cases, and with equal frequency in each lung; in the remainder of his subjects he found cavities of the size of an ordinary-sized apple or walnut.

HÆMORRHAGE IN PHTHISIS.

In early stages of phthisis, hæmorrhage is thought in many cases to occur in the bronchial tubes, without there being necessarily any destruction of lung tissue, as has been already mentioned (p. 445), but it is right to say that some authorities do not accept this explanation, holding that hæmorrhage in such cases is always indicative of the presence of tubercle. There can be no doubt that some escape of blood is very common indeed in the neighbourhood of tubercles, as may be seen in Fig. 110, and doubtless results from infiltration of the wall of the smaller vessels with tubercular growth. More considerable and sudden hæmorrhage must arise from rupture of a larger vessel, but this is extremely difficult to trace, it being sometimes impossible, even in cases of fatal hæmoptysis, to detect the injured vessel. It is generally clear, however, that the extravasation must have taken place into a cavity, or on a free surface, since the tissue is not lacerated as it would be were the hæmorrhage interstitial. In a certain number of cases of advanced phthisis, bleeding appears to result from the rupture of a small aneurism or aneurismal dilatation (see p. 389). Cases have been collected by Dr. Douglas Powell,* who gives the particulars of four cases which came under his own observation and of others from various sources, and draws the conclusion that—"fatal pulmonary hæmorrhage in cases of advanced phthisis almost invariably proceeds from rupture of a branch of the pulmonary artery in a cavity, either traversing its walls or crossing it, embedded in a tubercle." If blood, from whatever cause, be poured out into the bronchi, it may of course be drawn by inspiration into others than that in which it first appeared; and in this way solid casts are sometimes formed which are to be distinguished from those of plastic bronchitis.†

COMPLICATIONS OF PHTHISIS.

The complications of disease occurring in the course of tubercular phthisis and at different periods, are inflammations of the mucous membranes of the air passages of the pulmonary parenchyma and of the pleura. Of the appearances presented by the first, and of the relative frequency of occurrence in them of ulcerations in consumption, we have already spoken. There can be no doubt that the acrid character of the expectorated matters very much favours the ulcerative process. The frequency with which slight attacks of pneumonia supervene in the course of phthisis, is not surprising, if we look upon it merely as an exacerbation of the process actually constituting the disease. It appears in each case to cause an extension of the morbid process, and not to be the result of the previous elimination of tubercle. The pleurisy accompanying tubercle may supervene at various periods, and by the adhesions it causes and the consequent increased immobility of the lung, it necessarily much favours the development of the tubercular process. That the tubercular disease has a tendency to excite pleurisy, is evident from the frequency with which the latter occurs at the apices of the lungs, often forming a complete cartilaginoid cap from which it is difficult or impossible to detach the lung entire. When tubercular excavations approach close to the pleural surface, and are not preceded by thickening of the pleura, as generally occurs, a perforation may take place, inducing effusion of air and liquid into the cavity and secondary inflammation; we then have to deal with hydro-pneumothorax, upon which a rapidly fatal issue is almost certain to follow. Mere cellular adhesions, according to Rokitansky, cannot prevent this termination; they are, in part, mechanically loosened by the effusion from the cavern, and being involved in the pleuritic process, they are in part likewise destroyed in the exudation. Rokitansky describes three forms in which the communication between the tuberculous cavity and the sac of the pleura may be established; the pulmonary pleura may be detached from the affected surface by the mere force of the air rushing in so as to form a bulla, which afterwards bursts; it may be converted into a whitish eschar which tears or becomes detached unbroken, or the pleura, together with the infiltrated parenchyma surrounding the cavern, may become gangrenous and be converted into pulpy matter.

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* "Trans. Path. Soc.," vol. xxii. p. 41.

† Peacock: "Trans. Path. Soc.," vol. xxiv. p. 20.

The complication of pulmonary tubercle with tubercular disease in other organs is of very common occurrence; Louis* found tuberculous ulceration in the intestines in five-sixths of the cases he examined, the lymphatic glands were affected in the following order of frequency: the bronchial most, next the mesenteric, the axillary, mesocolic, lumbar, and cervical; the spleen and kidneys exhibited tubercular products in about one-sixth of the cases; several times the prostate was found more or less transformed into tuberculous matter, and tubercle was also met with in the cerebral arachnoid, though the frequency is not stated. The prevailing condition of the liver in pulmonary phthisis is one of fatty degeneration. Louis found it fatty in one-third of his cases. With regard to the co-existence of pulmonary tubercle, with tubercular disease in other organs, the general law has been established, that wherever, after the age of fifteen, tubercles present themselves in any organ of the body, we are certain also to meet with them in the lungs.

Obsolescence of Tubercle.—When the tubercular products in the lungs do not advance and undergo the progressive changes which we have described, a process of obsolescence occurs which appears to consist in certain chemical changes in the tubercular matter, followed by certain secondary alterations in the surrounding tissues by which they are adapted to the requirements of the case. These we find to exhibit two distinct kinds, which were formerly thought to depend upon a difference in the diathesis primarily giving rise to the disease. They are characterized by a fibrous or a calcareous metamorphosis. In the former case we find the tubercle assuming a more dense and leathery character, of a semi-cartilaginous consistency, drying or shrivelling up as it were, accompanied by a contraction of the superimposed tissue. If we examine the tissue containing tubercle under the microscope, we find mixed up with the ordinary forms of tubercle a distinct connective-tissue formation exhibiting a fibrous striated appearance. In the latter, a conversion of the tubercular matter seems to be effected into a chalky substance, at first moist and soft, gradually, owing to absorption of the fluid constituents, becoming harder and drier, and, at the same time, shrinking from its previous dimensions; thus, at times, we find, to use Hasse's words, "that a considerable portion of the lung, as may be inferred from the size of the bronchial tubes leading thither, becomes reduced to a hard shell, holding in its centre a chalky tubercle no bigger than a pea." The chemical characters of these formations have already been alluded to; they consist of amorphous carbonate and phosphate of lime, with, perhaps, other combinations of the same base; and much fatty matter. Lebert has also repeatedly observed cholesterine in chalky tubercles. The black pigment, which is at times met with to considerable amount in tuberculized lungs, and still more in the bronchial glands, does not present any different features from that commonly found in the pulmonary parenchyma. It appears to consist of pure carbon, and may present a mere amorphous granular form, imbedded in and scattered irregularly through the tissue, or it is found inclosed in an epithelial cell. The amount of the deposit is greater in proportion to the advance of life; and it has appeared to us to accumulate very rapidly in some cases of chronic inflammation. The presence of this black matter in the expectoration of phthisical patients in part gives rise to the greyish or dirty tinge we frequently observe. The question of the origin of this pigment has already been discussed.

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* "Researches on Phthisis," Syd. Soc. ed., p. 150.

Obsolete tubercle is surrounded by a dense capsule resulting from inflammatory induration. It is commonly found at the apices of the lungs, and may be easily felt on handling the part, and if near the surface their effect is rendered visible by a drawing in and puckering of a pleural surface. (Fig. 114.)

The metamorphosis of tubercle into fibrinous or cretified masses must be regarded as evidence of a curative tendency. Whether we are justified in assuming, as it appears Carswell* does, that an entire absorption of tubercular matter may take place without a metamorphosis of this kind, or without the formation of a cavity, is a question which we are not prepared to answer positively, though there is no certain proof to the contrary. That the healing process is not limited to the first stages of the disease, but is also seen after the formation of cavities, is established by the unanimous testimony of the best observers. Andral remarks that traces of cicatrization are found in individuals, who at one period of their life have been subject to a severe affection of the respiratory organs, which was regarded as phthisis, or in such as have been cured of a previous pulmonary attack, but have succumbed to a subsequent one of the same character, or, lastly, in persons who from the first day of their cough have continuously grown worse, in whom, therefore, after the cicatrization of one cavity new ones had formed. The cavities may disappear altogether, leaving a dense white fibrous tissue ramifying irregularly in the surrounding tissue, or the obliteration is incomplete, the cavity remains partially open, and the character of the lining membrane undergoes a change assimilating it to a serous membrane, or, which is more commonly the case, converting it into a vascular villous covering, resembling a mucous membrane. Rokitansky states that, in the latter case, aneurismal dilatation, or a gelatinous degeneration of the vessels subjacent to the membrane, is liable to give rise to hæmorrhage into the cavity, which either proves fatal, or else, by coagulating and plugging up the vessel, becomes a further means of obliteration and ultimate cure.

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* "Elementary Forms of Disease," art. "Tubercle."

Tubercle in Early Life.—Before quitting the subject of pulmonary tubercle, we have to allude to certain differences which exist between the manifestation of the disease in early life and later years. In the former instance the lungs are much less liable to become the seat of the disease than they are in the latter; it is more commonly simultaneously produced in a greater number of organs; and while, according to the extensive researches of Messrs. Rilliet and Barthez, which are confirmed by those of Dr. West, the "yellow form of tubercle" largely predominates, there is a remarkable immunity from tubercular cavities. The cavities that do occur are much smaller in proportion, and, though occasionally very numerous, do not give rise to the same amount of destruction of the pulmonary tissue that we see in the adult. The differences alluded to may be accounted for, partly by the greater share the nutritive organs take in all the functions of early life, and partly by the proclivity existing in the lungs to lobular inflammation. The latter circumstance renders it probable that a minute examination of tubercle in the infant, would exhibit a greater production of pus coincidently with the elimination of tubercular matter, than is found in the adult. We extract the following table from Dr. West's classical work on the diseases of childhood, as the best illustration that can be offered of the relation borne by tubercle to the different organs of the body in early and advanced life.

Of 100 instances in which tubercle was deposited in some of the viscera, it was present in

	Children from 1 to 15 years.	Adults from 20 years and upwards.	
	According to Rilliet and Barthez.	According to Louis.	According to Lombard.
The lungs	84	100	100
Bronchial glands . .	79	28	9
Mesenteric do. . . .	46	33	19
Small intestines . . .	42	33	0
Spleen	40	13	6
Pleura	34	2	1
Peritoneum	27	0	0
Liver	22	0	1
Large intestines . . .	19	10	0
Membranes of the brain	16	0	2
Kidneys.	15	2	1
Brain	11	0·8	2
Stomach	6	0	0
Heart and pericardium .	3	0	0

This table, as Dr. West remarks, shows not only that the liability of certain organs to become the seat of tubercle is different in childhood from what it is in the adult; but, also, that tubercle is simultaneously produced in a greater number of organs in the young than in the old.

IV.—FIBROID PHTHISIS.

Some authorities regard this as a distinct disease, others as merely ordinary phthisis which has lasted a long time, so that the caseous products below the tubercles have become absorbed or disappeared, while only the more prominent fibroid tissue remains; in fact, as "old phthisis." Others regard it as a different disease, *ab initio*, not necessarily connected with tubercle or with caseous inflammation. The question is too complicated to be discussed here. We can only state our belief in the occurrence of cases such as the following:—

1. Ordinary phthisis of both lungs, in which the fibroid change very greatly predominates, only a few tubercles or a little caseous matter being present to indicate the mixed character of the disease.

2. Extensive fibroid change in one or both lungs, with cavities, but with no tubercles visible to the naked eye, but where, nevertheless, they may be detected on microscopic examination.

Both these forms are fairly described as fibroid phthisis, and may occur in comparatively young persons (a little over thirty), so that age cannot be adduced as an explanation of their peculiarities. We regard them as extreme cases of ordinary phthisis, where one of the constant elements of this disease preponderates over the others.

3. Cases in which one lung only is affected with fibroid induration and partial destruction are more properly referred to the chronic pneumonia formerly described.

CANCER AND OTHER NEW GROWTHS.

No kind of tumour is common in the lungs as a primary growth, but most of them may occur secondarily when first produced in other parts of the body. Referring to the classification formerly given, we must begin with simple histioid tumours.

Fibrous tumours may be said generally not to occur in the lung either primarily or secondarily. One case is, however, recorded by Mr. Paget, in which a strictly fibrous tumour gave rise to secondary growths of the same kind in the lungs.*

The following have never been observed in the lung:—Myxoma, lipoma, angioma, adenoma, myoma.

Cartilaginous and bony tumours have both been observed as secondary growths after the recurrence or removal of similar tumours in distant parts. Instances of this metastasis of cartilaginous growths have already been given (p. 143). It has been supposed that enchondroma may occur in the lung as a primary growth, but this needs confirmation. The bony tumours found in the lung are generally, if not always, *osteoid.*

The form of simple tumour which most frequently occurs in the lung is *lymphoma.* Growths of this kind frequently originate in the bronchial glands or in the mediastinum, and may then spread by continuous infection into the lung. Many of these tumours have been described as cancer of the lung, or mediastinal cancer. The true character of the tumour, however, is apparent when a section of it is shaken up with water to remove the superabundant lymphoid corpuscles, when the characteristic stroma represented in Fig. 24 is apparent. Similar tumours may, however, originate in the mediastinal glands, and reach a very large size, without affecting the lung. The lung is attacked by a species of infiltration, which spreads peripherally from the root of the principal vessels and chief bronchi. Often the growth evidently takes place chiefly around the divisions of the pulmonary artery. Sometimes detached, isolated tumours are formed. The growth is usually pale, opaque, soft, and gives on section a very large quantity of creamy "juice," resembling, in fact, what was formerly regarded as the type of medullary cancer. Several cases of this affection have been described of late years,† and, doubtless, many more which were formerly described as medullary cancer really belong here.* It may, indeed, be taken as a well ascertained fact, that of the growths which arise in the anterior mediastinum or in the bronchial glands, some have all the infectious and destructive properties of what have been called, *par excellence*, malignant tumours, though preserving the lymphatic type of structure, and that these are extremely likely to affect the lung. The way in which they do so is clearly different from the secondary infection of the lung from a distant tumour.

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* "Surgical Pathology," 2nd edition, p. 762.

† "Trans. Path. Soc.," vol. xxiii. p. 245. 1872.

FIG. 117.

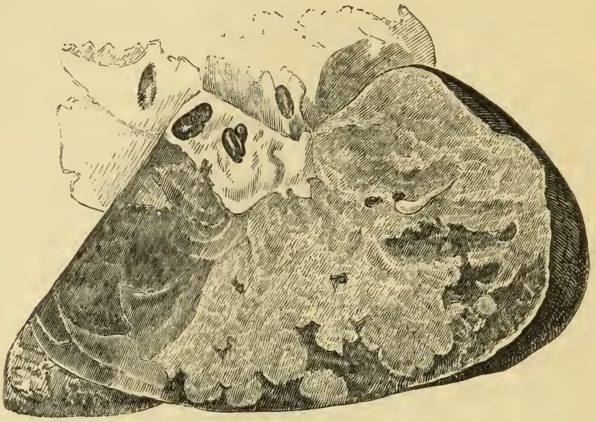
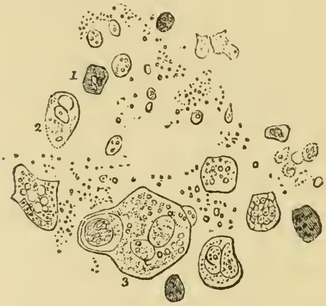


FIG. 118.



Infiltrated secondary cancer of the lung, with its microscopic elements. The lighter part is that containing the growth; it was of a brownish red tinge, and of greater density and hardness than the unaffected parts. It occurred in the right lung of a young woman, aged thirty-four, whose right bronchus was surrounded with a mass of medullary cancer, the right kidney also contained a large growth of the same kind, and the eleventh dorsal vertebra, especially its right side, was extensively destroyed by the same disease.

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* For instance, a case recorded by Dr. Wilks, in "Path. Trans.," vol. vi. p. 112, where a mediastinal growth produced infiltration of the lung, also affecting the heart.

Cancer of the Lung.—The variety of true cancer found most commonly in the lung is the medullary. If, however, the name be taken in the strict sense given to it in the first part of this work, it is not of frequent occurence; and is almost always secondary. Undoubted cases of primary cancer, though rare, have nevertheless been put on record. Dr. Walshe, in his work on Cancer, refers to a few; but with respect to some of them, it may be doubtful whether they were precisely what we now mean by cancer. Passing by earlier observations, we may refer to some in the "Transactions of the Pathological Society."*

The primary tumours are usually solitary, somewhat large masses; the secondary growths are sometimes in the form of multiple nodules, sometimes of an infiltration. Occasionally they seem to enter the lung from the pleura, on the surface of which they form flat plates or sheets. When projecting on the surface they are often cupped or umbilicated, like similar growths in the liver. The contraction of several such masses may produce a general puckered and fissured appearance like that of cirrhosis. Softening of the tumours may produce cavities which give the same physical signs as the vomicæ of phthisis, and broken down lung tissue with elastic fibres may under such circumstances be found in the expectoration. Other modes of growth of pulmonary cancer are as follows.† It may be prolonged from the pleural covering inwards by the interlobular septa, and thus divide the lung into a series of irregular polygons. It may also spread along the bronchial tubes, affecting first their exterior; next, the substance of the walls; and finally, the mucous membrane, along which it may extend almost indefinitely, forming, occasionally, casts like those of plastic bronchitis. Or again, it may implicate the vessels, especially the veins, and either occlude them, or grow into their cavity.

Sarcomata of the lung occur occasionally as secondary formations, subsequent to similar growths in other parts of the body; the spindle-celled form most frequently. Melanotic sarcoma is also found under similar circumstances. These growths form distinct tumours, not infiltrations, and grow in the walls of the alveoli, not within the air spaces. Dr. Wilks‡ speaks of having once seen a primary tumour of this character (fibrous or fibrocellular growth) in the lung. Ossifying sarcomata (one form of osteoid cancer) may also occur secondarily in the lung.§

Scirrhous cancer also occurs in the lung, most frequently as a consequence of primary scirrhus of the mamma. In such cases, it commences in the pleura.

Colloid has also occurred in the lung as a secondary growth.

Epithelioma of the lung is excessively rare. In a case reported by Dr. Moxon, it appeared to have been transported from the trachea into the bronchial tubes, and to have thus set up small secondary tumours.*

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* Bristowe, vol. xi. p. 34 (but perhaps a sarcoma). Fagge, vol. xviii. p. 29.

† Bristowe, "Trans. Path. Soc.," vol. xi. p. 25. 1860.

‡ "Trans. Path. Soc.," vol. ix. p. 31.

§ "Trans. Path. Soc.," vol. xx. p. 25. 1869.

SYPHILITIC DISEASE OF THE LUNG.

The morbid changes produced in the lung by syphilis are not as yet very well understood, except in certain peculiar and somewhat rare cases. As some syphilitic products seem much to resemble those of tubercle, they are, perhaps, sometimes mistaken for the latter. But on the other hand, as persons affected with constitutional syphilis often are very liable to tubercular phthisis, we must be cautious in referring destructive or degenerative changes met with in the lungs of such persons to syphilis alone.

The following are the chief forms of syphilitic disease met with in this situation :—

1. The true gumma, or syphiloma (p. 203).
2. A peculiar form of pneumonia, the "white pneumonia" of some authors, chiefly seen in congenital syphilis.
3. A species of indurative lobular pneumonia.

1. *Gummata of the lung* are rarely met with; and more frequently in congenital than in acquired syphilis; they have the characters already described in the first part of this work.

2. The *white pneumonia* of syphilis has been described by several authors. According to MM. Cornil and Ranvier, it occurs in newborn children, and up to the age of ten or twelve years. They regard it as interstitial pneumonia, and describe the interlobular connective tissue as in a state of rapid proliferation, presenting a large number of embryonic cells, and the interalveolar partition as thickened. The compressed alveoli are lined, or even filled with epithelial cells which are flattened when in contact with the walls, and rounded in the central space. The alveolar contents undergo fatty degeneration, and become absorbed, while the surrounding embryonic tissue becomes organized into fibrous tissue, so that a fibrous tumour, or a mass of fibrous material results, according as lesion was originally clearly defined or diffuse.

3. *Indurative lobular pneumonia* is the commonest syphilitic lesion of the lung in adults. It is often, both in its earliest and latest stages, confounded with tubercular phthisis, but in its middle stages is very different. It commences with minute granulations resembling broncho-pneumonic tubercle, which become confluent into larger nodules up to the size of a walnut or still larger. These masses are composed of very dense, engorged tissue, harder than ordinary pneumonia, of a dark red colour, mottled and paler in the centre. They are distinguished from tubercular or scrofulous products by a slighter tendency to degeneration. These

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* "Trans. Path. Soc.," vol. xx. p. 28. See also vol. xii. p. 46 of same series.

patches pass insensibly into the indurated and congested surrounding tissue. When degeneration sets in, the central portions become yellow, fatty, and necrotic, while the outer portions become more fibrous, and ultimately a mass of cheesy or calcareous material, or simply a fibrous cicatrix, is all that remains. The latter ultimate stages of degeneration are not unfrequently met with in the lungs of syphilitic individuals, and are hardly distinguishable from old tubercular products, except by being generally distributed through the lung and not occurring specially at the apex. It should be remembered that fibrous scars or puckered patches are less likely to be permanent in the lung than in more solid organs. The microscopical appearances are those of interstitial proliferation, with inflammatory changes in the air cells, ultimately resulting in the formation of rather dense fibronucleated tissue—*i.e.*, essentially what is described in the "white pneumonia." We have observed in one case a peculiar fibroid change round the pulmonary artery, suspected to be syphilitic.*

HYDATID AND OTHER CYSTS.

The formation of cysts in the lungs is of rare occurrence and perfectly latent, so that they are not discovered until after death, unless they excite irritation; they may then find their way into the bronchi and be expectorated. They occupy the lower lobes of the organs. They consist, themselves, of a double membrane of a clear pellucid appearance, which under the microscope presents an homogeneous, delicately laminated structure. The laminæ form parallel lines, so as to resemble the pages of an open book. The pulmonary tissue adjoining the cyst is covered by a dense membrane, so that, although entirely surrounded by the pulmonary parenchyma, there is not in reality any real intimate relation with it. They generally contain a limpid fluid, and present an endogenous development of hydatids of the same character as the parent cyst. They vary in size, but an instance which occurred to us, of an acephalo-cyst sufficiently capacious to contain a hen's egg, must be looked upon as unusually large. It neither contained secondary hydatids nor echinococci. A unique case of cysts in the lungs, filled with air, is quoted by Hasse.†

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* Payne, "Trans Path. Soc.," vol. xxv.

† "Pathological Anatomy," Syd. Soc. ed., p. 337.

CHAPTER XXXIV.

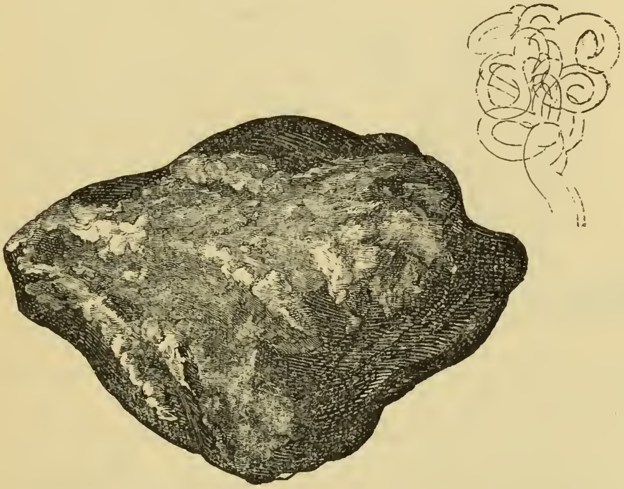
ABNORMAL CONDITIONS OF THE PLEURA.

PLEURITIS.

THE serous sac inclosing the lungs and serving to facilitate the movements of respiration, is more prone to morbid affections than any other serous membrane; of these, inflammation is the most frequent, and one that arrests the physician's attention very commonly both in the patient and in the dead subject. Some of the products of inflammation were formerly set down to physiological causes, owing to their being frequently met with in individuals whose histories did not give evidence of pleuritic inflammation having occurred in the course of their life. But the inference is not just, because even in severe pleurisies the symptoms are not necessarily of a character to attract the patient's attention, and most persons are familiar with the occurrence of occasional pains, of a not very enduring character, which may be accompanied by some effusion, though not of sufficient intensity to interfere with the function of respiration. The great frequency of the concurrent inflammation of the pulmonary tissue and its investing membrane, has given rise to a frequent misapplication of the term, and to a variety of theories in reference to the cause of pleurisy. So distinguished an author as Portal attempted to prove that pneumonia was not essentially different from pleuritis; but since the more careful prosecution of morbid anatomy, and the clearer distinction of symptoms during life which we owe to auscultation, no doubt exists that the two, though often associated, differ in their symptomatology as they do in their etiological and pathological relations.

The first stage of *acute* inflammation of the pleura is manifested by the appearances of greater or less congestion, causing a multitude of vessels not visible in the perfectly healthy pleura to the naked eye, to become filled with blood; a marked distinction may sometimes be observed between the venous and arterial channels, as exhibited in the different colours of the two systems. The vessels form an irregular network, and the more intense the inflammatory condition, the more uniform the redness becomes. At times we find the character of the congestion to be more punctiform, and to resemble, as Laennec has it, an attempt made to dot over the pleural surface with a paint-brush, with small spots of blood of an irregular shape, and closely approaching one another; it is probable that in many cases this appearance is due, not to the peculiarity of the disease, but to a partial emptying of some vessels as a post-mortem effect. The membrane, at the parts most affected, soon loses its natural transparency and gloss, in consequence of a secretion from the overcharged vessels investing its surface with a coating of lymph or fibrine, a straw-coloured, semi-gelatinous effusion which may be easily stripped off from the serous membrane. The microscopic appearances of this fibrine are delicate linear fibrillæ, of a generally parallel direction, enveloping and entangling the granular and corpuscular forms observed in fibrinous exudation. We very rarely meet with cases of what Andral has termed dry pleurisy, as the effusion is a rapid sequel of the first stages of inflammation; but we frequently have an opportunity of observing a limited production of lymph at one point, while the greater part of the remaining pleura or its fellow, may exhibit the first stage of the disease. This is especially the case on the pleural surface covering blocks of pyæmic or hæmorrhagic infarction. In the recent cases of inflammation occurring in healthy subjects, the effusion presents the appearance of a thin layer of thick cream, which, at the most dependent parts, seems to

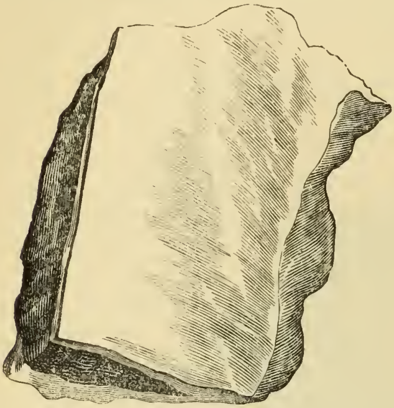
Fig. 119.



Straw-coloured lymph, coating the lower lobe of an inflamed lung, in recent pleurisy, before there was a trace of adhesion to the costal pleura. The outline represents one of two coils of new vessels, seen under the microscope in the fringe of lymph at the lower end.

be dropping from the organ. The older the effusion the more it assumes a membranous character, the friction and compression to which it is subject giving to it an irregularly honeycomb or cellular appearance, or causing it early to put on a filamentous or mossy form. The products of inflammation do not always present the characters just described, but vary much in cohesion, in colour, and quantity, according to the constitution of the individual attacked, and according to the exciting cause; thus we may, even in the same subject, meet with different products at different parts of the same lung. The exudation may be of a more serous or of a purulent character, in which case the sac will contain more or less of these fluids in which we find portions of lymph detached from the pulmonary surface floating about, while their peculiar colour, from an admixture of more or less blood, may present a proportionately reddish tinge. The more asthenic the type of the inflammation, the more the effusion departs from the character of a plastic exudation, the more inorganizable it becomes. The lymph or false membrane thus formed consists of a fibrinous reticulum and lymphoid corpuscles in variable proportion. The latter preponderate, as might be expected, in inflammations which take on the purulent type; the former in the more adhesive form of inflammation. The section which we have figured came from the surface of a pneumonic lung in the stage of purulent infiltration. As to the origin of these cells, of course, the same difference of opinion may prevail, as with respect to the new cells in all inflammatory processes. It has been clearly shown lately that there is a production of new cells, from the endothelium of serous membranes in the case of the peritoneum, which may by analogy be extended to the pleura. Cell-formation from proliferation of the connective tissue corpuscles has also been traced by Virchow and others. It is clear, from the figure, that young cells are contained in the interstices of the membrane, and not on its surface only. Emigration from the vessels may doubtless be also concerned.

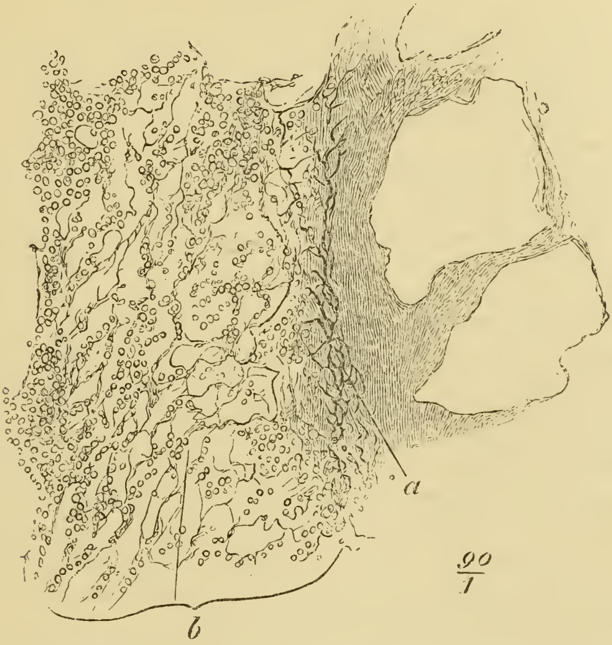
Fig. 120.



Portion of the lower lobe of the left lung of a patient, compressed by turbid serum occupying the pleural cavity. A thick layer of lymph covered the hepatized portion of lung; it was perfectly smooth from the contact with the liquid, and there was a free scalloped margin at some parts, of an inch in breadth.

When the progress of sthenic pleuritis is uninterrupted, the change that next ensues after the production of inflammatory lymph on the surface of the membrane, is the formation in the former of new vessels, and such further alterations in the exudation itself as to induce an assimilation to surrounding textures, and a restoration to a state of comparative, if not absolute,

Fig. 121.



Section through a pleuritic false membrane.

a. Elastic fibres of the original pleural tissue. *b.* Thickness of the false membrane, made up of a fibrinous network, with numerous leucocytes or pus corpuscles.

health. The adventitious membranes that thus become permanently formed are of greater or less extent, and may be limited to the one pleural surface, or connect the pulmonary and costal pleura, thus giving rise to further important changes of structure, which may seriously involve the entire thorax and consecutively even affect the spinal column. The adhesions are at first soft, easily separated, and vascular, but with time they assume an opaque, whitish hue, and become more firm the older they are. The great frequency with which they are met, has given rise to their being called ligaments of the lung, as if they formed a normal constituent of the organ.

The manner in which the new vessels, that we at a very early stage perceive in the exudation, are produced, has been the subject of much discussion, the arguments adduced being based upon physiological experiments or direct observations according to the inquirer's bias. The course of the new vessels is generally less tortuous and presents more parallelism than the vascular channels of the pleura; they appear to be active agents in effecting the absorption of a portion of the exudation matter, and after awhile a retrograde process ensues, and they in part cease to exist. The earliest trace of vessels which we have ourselves discovered in a case of acute pleuritic effusion occupying the base of one lung, where, as yet, no adhesions had taken place, and the creamy effusion was yet eminently fibrinous, exhibited the appearance of small coils, near the edge of the lymph, closely resembling a renal Malpighian tuft, into which two vessels could be seen entering; the sharp outlines of the formations, and a somewhat lighter tinge than the surrounding fibrine, alone marked them; they contained no blood corpuscles, and it was, therefore, only by inference that they were concluded to be young vascular channels. The thickness of the false membrane varies from a delicate film to many lines; it may itself become the seat of secondary inflammation, but in most cases it exerts a repulsive influence upon fresh attacks, and assists in protecting the adjacent parts from encroachment. A marked instance of this occurred under our observation lately, in an old man who had suffered a fracture of seven ribs of the left side, followed by inflammation of the corresponding pleura. The two surfaces of the upper half of the pleural sac had formed intimate adhesions, and the subjacent pulmonary parenchyma had remained in a healthy condition; the lower half contained two and a half pints of turbid serum, there was a thick layer of false membrane on the corresponding half of the lung, and this part of the organ was rendered unfit for respiration by the inflammatory process transmitted to it.

Accumulation of Fluid.—The amount of liquid effusion resulting from pleurisy varies from the smallest appreciable quantity to as much as twelve pints. A case in which this amount was removed from a man aged thirty-three, by paracentesis thoracis, is given by Dr. Novarra, in the "Medical Repository for 1820." Dr. Hodgkin* details an interesting case of empyema which partly discharged itself through the bronchi, and in which, after death, three large basins were filled with the sero-purulent contents of one pleural sac.

Liquid effusion not only necessarily induces compression of the lung on the affected side, pushing it upwards, but also displaces the adjoining viscera; the heart is forced over to the right thorax when the effusion is into the left pleural cavity; if into the right, the liver is depressed: in both cases the diaphragm is forced down, and its movements interfered with, while the intercostal spaces of the affected side exhibit a marked prominence, and the ribs are maintained in an elevated position. These anatomical features of extensive pleuritic effusion can scarcely be too strongly insisted upon in the bearing they have upon diagnosis. However, we must bear in mind that exceptional cases occur, in which, owing to a partial reabsorption of the fluid having been effected, the symptoms detailed are not always so marked as to render the diagnosis easy. Laennec observes, that at the epoch at which we ought to operate, the affected side, though full of pus, is less than the healthy side, owing to this circumstance, and the consequent falling in of the parietes.

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* "Lectures on the Morbid Anatomy, &c.," vol. i. p. 121.

Permanent Adhesions.—The secondary changes resulting from firm adhesion being formed, are of a different character. If they have been associated with previous extensive effusion, which has become absorbed, the degree to which the compressed lung will regain its former functions depends upon the duration of its confinement, and upon the firmness of the adhesions. The immediate consequence of the absorption was first shown by Laennec to be a falling-in of the affected side, owing to the expansion of the lung not taking place in the ratio of the removal of the liquid, and the firmer the adhesions are at given points, the more will this tendency be promoted by their increasing density and contraction. The depression is generally most marked at the lower part of the thorax, about the seventh and eighth ribs; and, owing to the consequent atrophy of the respiratory muscles of the affected side, the equilibrium is destroyed, and the spinal column is deprived of its symmetrical support; from this a curvature of the spine results, the convex margin of which is directed towards the healthy side. The shoulder of the diseased side sinks in proportion. A falling-in of the upper portion of the thorax, or of the infraclavicular region, is commonly noticed as an accompaniment of phthisis, consequent upon the formation of extensive cavities, and the coincident thickening and contraction of the pleura, to which we have alluded when speaking of tubercular disease of the lungs.

The pleural covering often becomes ultimately not only thick, but very dense, although in the early stages it will be found highly œdematous and almost gelatinous in appearance.

Pleural adhesions are so common as to be the rule in persons who have reached or passed middle life. They are not necessarily the result of inflammation properly so-called, but of the hyperplastic or proliferative processes which appear to be almost normal on serous surfaces.*

Time of Life.—The proclivity to pleuritis is greatest about the middle period of life, and diminishes in the ascending and descending scale of age. Before the fifth year it is not often met with. Dr. West observes that acute idiopathic pleurisy, unconnected with pneumonia, or in which the inflammation of the lung bears but a very small proportion to that of the pleura, is certainly an uncommon affection during the first years of childhood, and as a cause of death its rarity is extreme; and it certainly has appeared to us that, in cases of pneumonia, there is decidedly a less tendency in early life to excite pleuritic inflammation than we should observe in corresponding affections at a later period.

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* See Klein, "The Lymphatic System.' Part I. London, 1873.

With regard to chronic pleurisy, Dr. West remarks, "that while it is a very rare occurrence as a purely idiopathic affection in early life, it is one of the most common complications of the dropsy which often succeeds to scarlatina."

We do not generally find extensive pleurisy affecting both sides at the same time, while there is a marked difference in regard to the tendency of either pleura to inflammatory attacks; the left side presenting a much greater proclivity than the right. Hasse certainly states that the two sides are equally prone, but considers the fatality to be greater when the left side is attacked, than when the inflammation affects the right. The thirty-five fatal cases which he observed, were distributed in the following manner:—nine were double pleurisies, and in five out of the nine, the affection was trifling on one side; in the remaining twenty-six, the left side was the seat sixteen times, the right ten times. He also quotes Mohr's experience, who found that of fifty-six cases, the left side was the seat thirty-seven times, the right nineteen times. Doctors Hamilton Roe, Hughes, and Copland, are also of opinion that the disease exhibits a much greater frequency on the left than on the opposite side of the thorax. It appears from the observations of Messrs. Rilliet and Barthez that the converse is the prevailing character of infantile pleuritis, and that in children the right side is more liable to the idiopathic affection than the left.

Pleurisy in Phthisis.—There is no affection with which tubercular phthisis is so commonly associated as adhesions between the pleural surfaces, and the relation the two bear to one another appears to be in the ratio of the extent of the former. Hence the pleurisy has both a chronic and a more local character, and must be set down to the secondary irritation arising after the deposit has been effected. The gradual thickening assumes a cartilaginoid consistency, and the union becomes so intimate, that considerable force is often required to remove the lungs, and it is scarcely effected without laceration of their tissue. The intercurrent pleurisy accompanying tubercular disease of the lungs, is the source of those flying pains which, from time to time, attack phthisical subjects. The frequency of its occurrence is best illustrated by the statement of Louis, that in one hundred and twelve phthisical subjects, he found but one whose lungs were perfectly free, in every point of their surface. In eight cases only he found the right pleura wholly unattacked, and in seven the left; in these cases there were either no cavities in the non-adherent lung, or they were very small. Irritation, proceeding from other parts, may equally give rise to partial pleurisy; thus we find it limited to the diaphragmatic surface, in relation with a diseased liver, spleen, or peritoneum. Irritation of the mediastinal portion may be excited by morbid affections of the heart or bronchial glands; a limited effusion is frequently observed connecting the interlobar fissures, especially in connection with pneumonia and tubercular irritation.

EMPYEMA.

This is the term applied to a collection of pus in the pleural sac, produced by inflammation, which may be the result of acute or of chronic disease, but is specially likely to occur in cachectic subjects, or those predisposed to the chronic inflammations called scrofulous. Whether this is usually preceded by an effusion of serum is uncertain, since physical examination does not enable us to discriminate between the two. When the pleural cavity is found full of pus, the surfaces are either simply covered with false membranes of inflammatory lymph, or else, in more chronic cases, the membranes are greatly thickened, and resemble the so-called pyogenic membrane which lines an abscess. The pus is seldom or never so thick as that of an abscess proper, being rather a purulent serum. It is impossible to regard it as anything else than the result of inflammation.

A marked difference exists between the pleura and peritoneum in regard to this point, for, while the former is peculiarly liable to effusions resulting from a low form of inflammation, the fluid accumulations that we meet with in the latter, are more often of a mechanical origin; hence the result of operative interference is very much more favourable in cases of empyema and inflammatory hydrothorax than in ascites; in the former, when the diseased condition giving rise to the effusion has subsided, there does not exist a tendency to repeated accumulation as in the latter; hence paracentesis thoracis is more likely to prove a curative agent than tapping of the abdomen, where it rarely serves otherwise than as a means of palliating urgent symptoms. Dr. Hamilton Roe, who has disproved Laennec's statement, that paracentesis was rarely successful, obtained a recovery in eight out of nine cases of empyema, and of nine out of thirteen of inflammatory hydrothorax; and in the same paper from which we derive this information,* we find that Mr. B. Philips records a brief analysis of 122 cases of paracentesis, thirty-one of which were performed for empyema, and nine for hydrothorax; of the former twenty-six, of the latter six were cured.

It is, however, admitted on all hands, that it is essential to the success of paracentesis thoracis that it be performed at an early period of the disease.

When the fluid is not evacuated by an operation it is occasionally discharged spontaneously, either by perforation of the pulmonary tissue, by the thoracic parietes, or by the diaphragm. Dr. Williams considers the second, while Laennec and Hasse look upon the first as the more frequent occurrence. The perforation of the intercostal spaces takes place, not as would be expected, at the base of the lung, but about the middle lobe of the lung; the external discharge being generally effected by sinuous openings burrowing under the integuments. The prospect of recovery is greater here than when perforation of the pulmonary pleura leads to an effusion into the lung, for, in the latter instance, in addition to the mechanical influence of the fluid filling the bronchi, we have to deal with the contamination of the system likely to result from the decomposition of the fluid, produced by its contact with the atmosphere.

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* "Medico-Chirurgical Transactions," vol. xxvii.

It is often said that pus in the pleura never becomes absorbed, but the following appearances, occasionally met with, seem to result from a partially healed empyema. A collection of clear amber-coloured fluid, enclosed by very thick fibrinous walls, sometimes in very numerous layers, usually at the base of the lung, or behind, with evidence of great contraction having taken place. These are precisely the appearances of an abscess where no evacuation has taken place, but where absorption is nearly complete.

PNEUMOTHORAX.

Perforation of the pulmonary tissue by empyema is one of the modes in which an accumulation of air in the pleural cavity is produced. The most frequent origin of this condition is perforation of the pulmonary pleura by the extension of a tubercular cavity, before the opposing surfaces have become agglutinated by fibrine; the mere softening of one or two tubercles formed close to the pleura, and communicating with a minute bronchus, is described by Dr. Copland as another, though rarer cause of this accident. It has been found to result from rupture of emphysematous vesicles; and Rokitansky also states that it may be consequent upon perforation of the diaphragm or of the mediastinum, arising from acute softening of the stomach or œsophagus. That perforation of the superficial parts leading to the pleural sac induces pneumothorax, will be naturally inferred at once; but it is more than improbable that it is ever due to the evolution of gases from the fluids of hydrothorax by spontaneous decomposition, until after death. The perforation leading into the pleural sac is generally a small oval aperture, or a mere fissure, a few lines in length, and situated in the vicinity of the third and fourth rib near the axilla. The left side offers the greatest liability; Louis found it affected in seven out of eight cases; Hasse has met with nine in which the left, and seven in which the right side was the seat of the lesion; and of fifty collected by Reynaud, thirty-three were on the left and seventeen on the right side. The immediate result of the perforation is imminent if not actual suffocation from collapse of the lung; if death does not at once ensue intense pleuritis is set up, and the patient rarely survives for many days. We find the lung compressed to the utmost, and the other viscera are much displaced, according to the side in which the air has accumulated, the epigastrium protruding from the descent of the diaphragm, and its action on the liver and stomach: the other pathological conditions are those indicating inflammation of the pleura; and we may observe an attempt at repair in the shape of a false membrane investing the fissure. The occurrence of pneumothorax is only possible when the pleura at the point of perforation has not previously become adherent; but it sometimes happens that the ulcerative process is continued after the pulmonary and parietal pleura have been agglutinated, and passing through the uniting medium attacks the intercostal muscles and integuments. A fistulous opening may thus be established.

HYDROTHORAX.

Hydrothorax, or dropsical accumulation of fluid in the pleural sac, occurs in two forms, as a primary and as a consecutive lesion; the former is a disease of much less frequent occurrence than was at one time supposed. Laennec states that one could not establish a higher ratio for the occurrence of idiopathic hydrothorax as a cause of death, than one in two thousand. Let us hope that the days are past in which such errors of diagnosis as he alludes to can be committed; for he asserts having found hypertrophy of the heart, aortic aneurism, phthisis pulmonalis of a somewhat irregular character, and even scirrhus of the stomach or liver, without the least effusion into the pleura, mistaken for hydrothorax. The affection consists in the effusion of a limpid serosity into the pleura, to a greater or less amount, generally limited to one side, and unaccompanied by any appreciable change of structure in the serous membrane; the compression exerted by the fluid upon the lung and the adjacent parts is necessarily the same in this instance as in those forms of fluid accumulation which have already been considered.

The secondary or symptomatic form of hydrothorax is a common sequel of acute or chronic diseases, heralding the fatal termination, and giving evidence of that loss of tonicity that results from exhaustive maladies. The circulating system is more frequently found to be at fault than any other; hypertrophy of the heart, valvular disease, pericarditis, are common causes; it is also often associated with renal degenerations, tubercular and cancerous affections. The same cause that induces the effusion into the pleura, gives rise to dropsical accumulation in other serous cavities, or in the cellular tissue; hence symptomatic hydrothorax is generally accompanied by other affections of the same kind. As in the primary form, we find no definite lesion of the serous surface associated with it; it is not in fact an affection of the membrane at all, but exclusively of the vascular system, and we must look to the blood and the capillaries for an explanation. According to Laennec, it rarely occurs more than a few days before death; and though it often produces no sensible effect upon the patient's feelings, it often causes suffocative attacks, which render his last moments painful and distressing. The liquid itself is commonly a clear straw-coloured serum.

HÆMOTHORAX.

When the pleural sac is filled with blood, or with a fluid of a decidedly sanguineous character, we have the condition known as hæmothorax. This is commonly due to some mechanical lesion, or to the rupture of an aneurism; but it is also said to be met with as the result of capillary exhalation—of the same character as that to which active or passive spontaneous hæmorrhages are commonly attributable. It is said to be capable of reabsorption, or to superinduce inflammatory action; or again, to be liable to decomposition, and thus to give rise to pneumothorax.

GANGRENE.

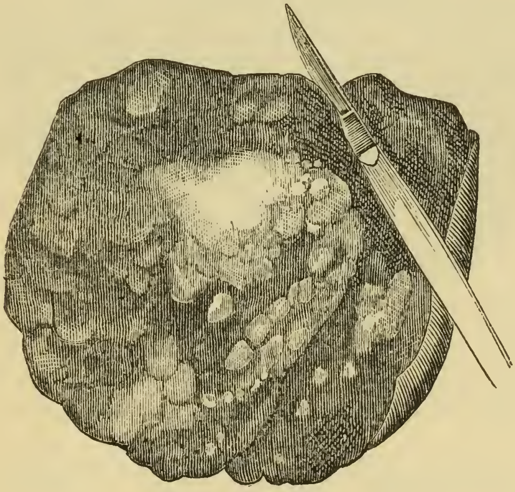
Before considering the adventitious products met with in the pleura, we must briefly advert to the occurrence of gangrene. It is an unusual lesion, and is commonly connected with gangrene of the pulmonary tissue. The sloughs are to be recognized by their greenish, brown, or blackish hue, of a circular or irregular form, extending to some distance beyond the part detached. The fœtid smell will also assist in determining the character of the lesion.

ADVENTITIOUS PRODUCTS.

Among the homologous formations occurring in the serous membrane of the lungs, authors enumerate cartilage, bone, and fat. A cartilaginoid thickening of the pleura, more especially at and about the apices, is by no means unusual; but the microscope invariably resolves this deposit into one of a fibrous, or as Lebert terms it, chondroid, character. If the result of gone-by inflammation of the pleura, it affects the free surfaces of the membrane, with which it intimately coalesces; when due to subserous congestion, it is found in the subserous tissues, and in the membrane itself. The lung exhibits fibroid formations, which, like those found on the heart, the liver, or the spleen, are frequently but the indications of a chronic hypertrophic process, independent of any actual inflammation; they are smoothed, nodulated, very dense, and adherent only by their exterior surface. We meet with the formation of true bone in the pleura as little as of genuine cartilage:

the osteoid deposits are mere amorphous aggregations of calcareous matter, occurring in plates or irregular points: they may be encysted, and occasionally they form pedunculated projections invested by the pleura; to this class we may probably refer the case of ossification of the lungs given by Dr. Baillie,* for, in the delineation, the ossified parts are distinct polypoid off-sets from the pulmonary tissue, though apparently invested by the same membrane. Rokitansky states that fibrous thickenings may occur on the costal as well as the pulmonary, but that they only ossify on the costal pleura, the subserous osteoid products occurring chiefly in the intercostal spaces, from which they may be discharged into the cavity of the thorax in the shape of round nodules. That ossification of the pleura is generally preceded by some inflammatory condition, may be inferred both from the frequent occurrence of other inflammatory products in the pleura, and from the analysis of a considerable number of cases in a dissertation by Dr. Posselt;† he found that out of twenty-seven instances twelve affected the right and fifteen the left side, while the ratio of the sexes was as thirty men to four women. The size of the osteoid deposit is occasionally very extensive—Dr. Hodgkin removed from an old man who died at Guy's Hospital, a plate of bone subjacent to the parietal pleura, which half encircled the chest, and formed a con-

FIG. 122.



Old cartilaginoid capsule of the apex of a lung, in a man aged sixty-three; both lungs were similarly affected, and like patches were also found on the spleen. There was some appearance of obsolete tubercle, and much black pigmentary matter. No definite structure was to be traced in the capsule by the microscope.

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* "Morbid Anatomy," second fascic. pl. vi.

† "De Pleuræ Ossificatione." Heidelberg, 1839.

siderable mass. The fatty deposits which we find connected with the pleura are not seen on the free surface, but seem to be the result either of a transformation of previous inflammatory products, or a normal fatty growth under the serous membrane. Recent fibrinous exudation occasionally closely resembles adipose tissue both in colour and form, though there can be no real difficulty in determining its nature.

Tubercle is not very frequently met with in the pleura; and almost exclusively occurs as a secondary form of the disease. The pleura in this respect differs in a marked manner from the peritoneum and the arachnoid, both of which are more prone to primary tuberculosis than the former. Tubercle forms either under the pleura or on the surface. Tubercles of the latter description, Dr. Hodgkin remarks, when closely set, are associated with thickening of the membrane, and may form almost continuous sheets. This is seen chiefly in some of the kinds of general tuberculosis, when other serous membranes, or

Fig. 123.

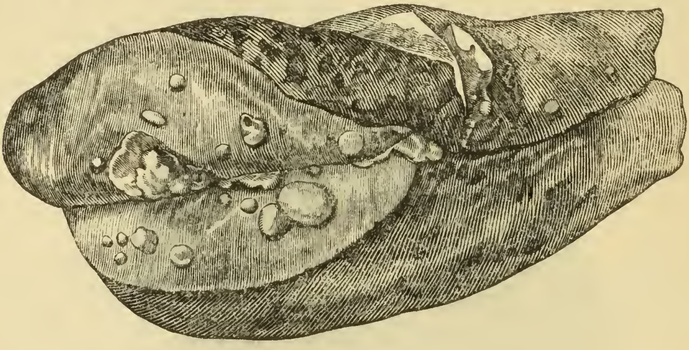
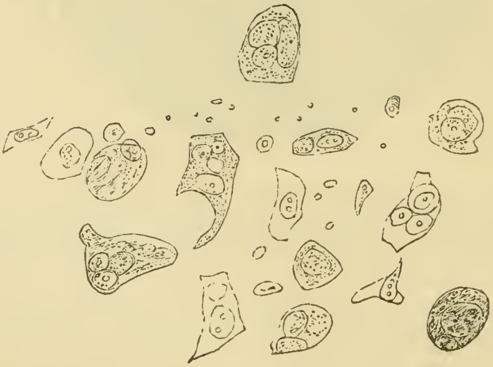


Fig. 124.



Naked eye and microscopic views of cancer of the pleura. The growths were mainly in the interlobar fissures, and occurred in a female who had malignant disease of the left mamma. The liver also exhibited cancer nodes.

the lungs, will probably be simultaneously affected. The lungs are in these cases probably never affected without some participation of the pleuræ. In more chronic cases the tubercles are found chiefly in thickened pleuræ, or even, according to Rokitansky, in actual false membranes. The highly vascular œdematous adhesions met with in chronic phthisis sometimes furnish the most exquisite examples of early stages of tubercle, which will be found following the course of the blood-vessels. Rokitansky again describes tubercular abscesses which form in pseudo-membranous structures, and may perforate the pleura, and even the thoracic walls.

Malignant growths never affect the pleura primarily, but involve the membrane by extension from the mamma, the bronchial glands, the mediastina, or from other neighbouring tissues. They appear on the pleura as flattened masses, rarely larger than an almond, surrounded by a halo of blood-vessels. While tubercle occurs in very numerous small spots spread all over the membrane, cancer is only seen in a few isolated patches. Both are liable to induce serous effusion of a sanguinolent character. Medullary carcinoma and melanotic cancer are the forms of malignant disease that most frequently attack the pleura.

Hydatid cysts appear to occur in the pleura. Cruveilhier[*] details a case in which a large number of acephalo-cysts were discharged from an artificial opening, apparently communicating with the pleural cavity, in a whitesmith aged twenty-nine; the man entirely recovered, after the evacuation of above five hundred hydatids. Dr. Hodgkin,[†] in his fifth lecture, speaks of a specimen presented to Guy's Museum, in which a large cyst containing acephalo-cyst hydatids is situated in part beneath the close pericardium about the base of the heart, and partly under the pleura pulmonalis at the root and summit of the right lung.

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[*] "Anatomie Pathologique," vol. i. p. 247.

[†] "Morbid Anatomy of the Serous Membranes," vol. i. p. 137.

THE PATHOLOGICAL ANATOMY OF THE ALIMENTARY CANAL.

CHAPTER XXXV.

I. OF THE MOUTH AND FAUCES.

Congenital malformations of the lips and cheeks are usually associated with defects of the bony structures. Most of these malformations are of the nature of defective or arrested development; the parts being imperfectly formed, or stopping short at a lower stage of development. The following varieties are enumerated by Förster.*

1. *Agnathia*, or deficiency of the lower jaw. This is combined with imperfect development of the upper jaw. There is, properly speaking, no lower part of the face, and the mouth is either absent or closed posteriorly. This defect is incompatible with life; it is very rare.

2. *Prosoposchisis*, or fissure of the face. The face remains, as in its earliest stage of development, one large fissure; the buccal and nasal cavities are usually continuous; the eyes frequently defective and the orbits involved in the large fissure. This defect is incompatible with life; it is not so very rare.

3. *Cheilo-gnatho-palato-schisis:* Cleft palate, and Hare lip. A fissure exists in the upper lip, the upper jaw and the palate depending on imperfect union of the superior maxillary bones with the intermaxillaries, or of the palatine processes with one another. Fissure of the lip may exist alone; it is then either unilateral, bilateral, or median, the latter condition arising from confluence of two lateral fissures. The gap is always between the external incisor and the canine teeth. Fissure of the jaw does not occur without fissure of the lip. It passes through the corresponding part of the superior maxillary bone, and sometimes reaches the nose. It is usually bilateral, but the intermediate part is sometimes wanting.

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* "Handbuch der Path. Anat.," vol. ii. p. 1; "Die Missbildungen des Menschen," pl. xxiv., xxv.

Fissure of the palate may be unilateral or bilateral, and combined or not with fissure of lip and jaw bone: it may affect the hard or more often the soft palate alone, or both. That of the hard palate is on one or both sides of the vomer, never in the median line; that of the soft palate always median. Some degree of this defect is not uncommon, and quite compatible with life. A similar fissure of the lower lip and lower jaw is very rare.

4. *Mikrognathia*, or *Brachygnathia*, deficient development or small size of the jaws, is a very rare defect, usually affecting the lower jaw.

5. *Synchelia*, or *Atresia oris*, complete closure of the mouth by adhesion of the lips; or a very small opening may be left.

6. *Mikrostomia*, smallness of the mouth, which is formed by small, but naturally-disposed lips.

7. *Achelia*, *Mikrochelia*, deficiency or small size of the lips, which expose the teeth or gums.

8. *Makrostomia*, prolongation of the corners of the mouth on one or both sides through the cheeks in the direction of the ears.

INFLAMMATION.

The buccal mucous membrane shows but little tendency to be affected by catarrhal inflammation; it is, however, often inflamed in one or more spots from some local irritation, or morbid action. Thus a carious tooth, a piece of diseased bone, a crop of herpetic vesicles, will excite inflammation in their vicinity. In adults, general inflammation of the mucous membrane is sometimes produced by the abuse or excessive action of mercury, and shows a marked tendency to pass into a state of ulceration and sloughing. Nitrate of silver, iodide of potassium, and probably other drugs, may have a similar effect.

Mr. Tomes mentions the occasional occurrence of spontaneous salivation, with considerable inflammation of the gums. Chronic inflammation of the gums is not uncommon, and has appeared to us sometimes to be of a rheumatic origin. It may extend over the whole mouth, or be confined to the vicinity of two or three teeth. "The surface of the gums," Mr. Tomes says, "becomes minutely nodulated; and the secretion of epithelium increased; the papillæ are increased in prominence, while the substance of the gum is generally thickened, and the edges about the teeth become thick and round."

In another form of so-called chronic inflammation, the gum rather decreases in size, and "assumes a very smooth and polished surface and mottled aspect:" the hard palate also becomes implicated, and there is acute intermittent pain. Ulcerations often form on the gums as well as on other parts of the mucous membrane of the buccal cavity; they are sometimes simple aphthæ, sometimes small, round, slightly excavated, and without any surrounding inflammation. In some cases the ulcerations are attended with much inflammation and swelling of the mucous membrane and subjacent tissues, and in others they are rather of a sloughy nature, and form upon a surface dark coloured by asthenic congestion.

TUMOURS OF THE JAW.

The term *Epulis** has been generally but loosely used for any tumour of the gums not supposed to be cancerous. There are several different forms. One form of Epulis is a fibrous tumour which originates in the fibrous tissue of the gums, or in the periosteum, and not only grows outward towards the cavity of the mouth, but also penetrates into the Haversian canals and cancelli of the bone. Its surface is generally pretty smooth, "like the gum," or it may be rough and more or less lobulated. "Osseous spiculæ, not uncommonly shoot," according to Mr. Tomes, "from the jaw into the tumour, and in some cases isolated nodules of bone are found in the substance." The last-mentioned fact we have several times observed. Spindle-celled sarcoma or "recurrent fibroid" has also been described under the same name; and again specimens of Sir J. Paget's "myeloid" tumours come under this head. In all these cases (as pointed out by Mr. Hawkins) the growth springs from the bone or periosteum.

Polypus of the gum is a local hypertrophy of its tissue, sometimes occasioned by mechanical irritation. It shows, "on section, fibro-cellular tissue, covered by a thick layer of epithelium," and like polypi of mucous surfaces in general, is distinguished by the abundance of glands. Cysts may also form in the tumour.

Vascular tumours, or *Angiomata*, consisting essentially of dilated vessels, sometimes appear on the gums. Mr. Tomes describes one of a bright scarlet colour, soft in texture, and easily compressed and emptied of blood, and prone to bleed on slight irritation.

Cancer, almost always in the form of scirrhus, occasionally attacks the gums. Its size varies usually from that of a pea to that of a nut. It ulcerates after a time and may throw out fungous growths.

The vesicles of herpes and the pustules of variola are occasionally developed upon the buccal mucous membrane.

CROUPOUS AND DIPHTHERITIC INFLAMMATION.

These two processes are by no means clearly distinguished when they affect the mouth. According to Förster they are usually found successively in the same case, the simple croupous process passing into a necrotic or gangrenous form, which is diphtheria. Ordinary croup or stomatitis pseudo-membranacea is characterized by redness and swelling of the mucous membrane, followed by the formation (often very rapid) of yellowish or ash-coloured false membranes, which may attain a considerable thickness in the angle between the gums and the cheeks, or under the tongue. At first they are firmly attached, but soon become looser, and when removed, leave an injected, but not ulcerated surface. This may occur in limited patches or over a wide area. It may stop short at this stage or go on to a form more resembling diphtheria. This disease may, it is said, be produced by local irritation, but is more commonly an extension of pharyngeal croup, or else the accompaniment of some specific disease, such as measles, scarlatina, erysipelas, &c. True diphtheria of the mouth is probably always a manifestation of the constitutional infectious disease thus named, and may accordingly be transmitted by direct contagion. In its early stages it resembles the process just described, but the false membranes when removed, leave an excoriated surface which may be decidedly ulcerated, the whole epithelium being destroyed. The colour of the exudation, which is naturally whitish, is often rendered darker by sanguineous effusion saturating it; when this is the case its aspect, and the extreme fœtor which it exhales, give to it a considerable resemblance to a gangrenous slough. This resemblance is increased if the exudation be situated, as it sometimes is, beneath, and not upon the layer of epithelium.

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* Etym., ἐπὶ = upon, οὖλον = the gums.

In such cases the process extending more deeply causes necrosis or gangrenous destruction of the whole thickness of the mucous membrane, and even submucous tissue. The gums themselves ultimately degenerate into a bad-looking, pulpy, sanious mass, and the mucous membrane of the cheeks and fauces, underneath the exudations, is equally found converted into a friable, fœtid pulp, or a firm slough. The slough which separates then consists of necrotic tissue as well as false membrane; and in some cases, as in diphtheria of the throat, deeper parts, such as periosteum and bone, may be affected. If recovery take place, formidable scars are left, which may cause much injury by their contraction. Actual destructive or necrotic processes are in fact, as before stated, in speaking of similar affections of the throat, the only positive anatomical marks of diphtheria. Vegetable parasites, such as oïdium albicans, are often found in the gangrenous masses.

Of the clinical features of the disease, we can only say that the epidemic adynamic character of the above described process is now well known. Andral, writing more than twenty years ago, contended most justly that the congestion of the part affected, though first in order, was but secondary in regard to causal agency; and the disease is now always classed among general or constitutional diseases. Andral mentions that blistered and all denuded surfaces, during the prevalence of such epidemics, become covered with false membranes like those which form on mucous surfaces. Wounds and ulcers at such periods are found unusually inapt to heal, and all the experience of the *juvantia* and *lædentia*, seems to testify that the local morbid process is extremely different from common inflammation, and is essentially dependent upon a grave alteration of the general system.

ULCERATION OF THE MOUTH.

Aphthæ, Thrush, Muguet.—*Aphthæ* are small whity specks, sometimes so closely set together that they coalesce and form patches, which may be very extensive. They separate after a time, leaving the mucous membrane beneath either simply excoriated or superficially ulcerated. After being detached they are often produced again, and this may occur several times in succession. The true nature of this disease has been the occasion of much controversy, and there is not yet a perfect agreement on the subject. The patches consist, there is no doubt, chiefly of clusters of the mycelium and spores of a microscopic fungus, the oïdium albicans, found very frequently in small quantities in the mouth even in health, and more abundantly in inflammatory or diphtheritic processes. It is only in the disease now under consideration that the fungus forms nearly the whole of the adventitious mass, which contains beside only a few detached epithelial scales, beset with spores. It may thus be described as a growth of fungus upon the mucous surface. It has also been shown that it may be transferred by transplantation to the mouths of healthy children. It will not, however, flourish unless there be some unhealthy state of the mucous membrane, and acidity appears to be the most important favouring condition. The mucous surface of the mouth appears to be in new-born children always acid, even in health—a circumstance which accounts for the facility with which the disease develops itself at that age; while in older children, a very slight disturbance of nutrition serves to bring about the conditions necessary for its development. There is no doubt that the most frequent occasional cause of aphthæ is the fermentation of portions of milk which remain in recesses of the mouth. It should be mentioned that the term *muguet* is applied by the French to the more extensive deposits of this kind. Bad health, indigestion, or abdominal disorders, are the precursors and attendants on aphthæ. The whole of the buccal mucous membrane appears to be in a state of asthenic inflammation, and the same condition extends in some measure to the whole alimentary tract. Adults are sometimes affected by aphthæ as the result of indigestion, or as indication of decaying vital powers. A late eminent physician prognosticated his own approaching decease from the appearance of aphthæ on his tongue.

Dr. H. Salter describes small circular ulcers which form at the tip and along the edges of the tongue. These we have experienced ourselves, and can scarce think they should be separated from aphthæ. According to him, they are produced by the effusion of lymph into one of the fungiform papillæ, which soon disappears by sloughing or ulceration, leaving an ulcer which continues to spread for some time.

Follicular Stomatitis is described by Dr. West as sometimes idiopathic, sometimes a concomitant of measles. In either case it is rare, after five years of age. "The mouth is hot; its mucous membrane generally of a livid red, while a coat of thin mucus covers the centre of the tongue. On the surface of the tongue, especially near its tip on the inside of the lips, the cheeks, near the angles of the mouth, and less often in other situations, also, may be seen several small, isolated, transparent vesicles, or the ulcers, which, after bursting, they leave behind. The ulcers are small, of a rounded or oval form, not very deep, but having sharply cut edges; and their surface is covered by a yellowish white, firmly-adherent slough." "When the ulcers are healing, no change in their aspect is observable, and they continue to the last covered by the same yellow slough, but, by degrees, they diminish in size; and seldom or never is any cicatrix observable in the situation which they occupied." The vesicles form in crops, not generally containing many; the resulting ulcers sometimes coalesce and form a continuous patch. The affection is sometimes complicated with herpes of the skin of the lips, and might almost be considered as a similar eruption of the mucous surface.

Ulcerative Stomatitis, or Gingivitis.—This disease which commences in the gums, is thus described by the same observer: "The gums are red, swollen, and spongy, and their edge is covered with a dirty white or greyish pultaceous deposit; on removing this their surface is exposed, raw and bleeding. At first only the front of the gum is thus affected; but as the disease advances, it creeps round the teeth to their posterior surface, and then destroying the gum, both in front and behind them, leaves the teeth denuded, and very loose in their sockets. On those parts of the lips and cheeks, however, which are opposite to, and consequently in contact with, the ulcerated gums, irregular ulcerations form, which are covered with a pultaceous pseudo-membranous deposit, similar to that which exists on the gums themselves. Sometimes, too, deposits of false membranes take place on other parts of the inside of the mouth, the surface beneath being red, spongy, and bleeding, though not distinctly ulcerated. . . . When recovery has commenced, the disease ceases to spread; the drivelling of fœtid saliva diminishes, the white, pultaceous deposit on the gums, or on the ulcerations of the cheek [illegible] lips, becomes less abundant; the ulcers themselves grow less; and, finally, the gums become firm," and slowly, and perhaps with partial relapses, regain their healthy condition. The disease is common, rarely fatal, rarely associated with, or proceeding to, gangrene.

Gangrene of the Mouth; Noma; Cancer aquaticus; Cancrum Oris.—True gangrene of the mouth is a much less frequent, and much more fatal affection. It is very seldom idiopathic, almost always occurring consecutively to measles or some other disease. Messrs. Rilliet and Barthez found, out of twenty-nine cases, nineteen aged from two to five years, and ten aged from six to twelve. We again quote Dr. West's description, which pictures very well the only case which we have witnessed ourselves. There is at first scarce any suffering, and some unusual fœtor of the breath, some profuse secretion of offensive saliva, and swelling of the cheek, are the first circumstances which are observed. The characters of the swelling of the cheek are almost pathognomonic. It is not a mere puffiness, but is tense, red, and shining—looking "as if its surface had been besmeared with oil, and in the centre of the swollen part there is generally a spot of a brighter red than that around. The cheek feels hard, and is often so unyielding, that the mouth cannot be opened wide enough to get a good view of its interior. The disease is almost always limited to one side, and generally to one cheek." Occasionally it begins in the lower lip, never in the upper, but it may extend to either. "Whatever be the situation of the external swelling, there will generally be found within the mouth, at a point corresponding to the bright red central spot, a deep excavated ulcer, with irregular jagged edges, and a surface covered by a dark brown, shreddy slough. The gums opposite to the ulcer are of a dark colour, covered with the putrilage from its surface, and in part destroyed, leaving the teeth loose, and the alveoli denuded. Sometimes, especially if the disease be further advanced, no single spot of ulceration is recognizable, but the whole inside of the cheek is occupied by a dirty putrilage, in the midst of which large shreds of dead mucous membrane hang down. As the disease extends within the cheek, a similar process of destruction goes on upon the gum, and the loosened teeth drop out one by one. The saliva continues to be secreted profusely, but shows by the changes which take place in its character the progress of the disease. At first, though remarkable for its fœtor, it is otherwise unaltered, but afterwards loses its transparency, and receives from the putrefying tissues, over which it passes, a dirty, greenish, or brownish colour, and at the same time acquires a still more repulsive odour. While the gangrene is thus going on inside the mouth, changes no less remarkable are taking place on the exterior of the face. The redness and swelling of the cheek extend, and the deep red central spot grows larger. A black point appears in its midst, at first it is but a speck, but it increases rapidly, still retaining a circular form —it attains the bigness of a sixpence, a shilling, a half-crown, or even a larger size. A ring of intense redness now encircles it, the gangrene ceases to extend, and the slough begins to separate. Death often takes place before the detachment of the eschar is complete; and it is fortunate when it does so, for sloughing usually commences in the parts left behind. The interior of the mouth is now exposed, its mucous membrane and the substance of the cheek hang down in shreds from amidst a blackening mass," which exhales a horrible fœtor. There is no acute pain throughout, the patient is generally rather drowsy, and death takes place quietly in most cases. No cause has been assigned for the occurrence of gangrene in this part, all that can be said is that a true mortification or death of the textures seems to take place, which is itself the primary evil, and not the result of inflammation, disease of the vessels, or obstruction of their channels. This is a good illustration of the doctrine we maintained, when speaking of mortification, viz., that it essentially consisted in a loss of the vital powers which maintain, in opposition to those of inorganic chemistry, the complex constitution of the animal tissues.

MORBID CONDITIONS OF THE TONGUE.

Macroglossia, Hypertrophy of the Tongue.—The tongue is liable to be affected by an extraordinary hypertrophic enlargement, in consequence of which it protrudes from the mouth, sometimes as much as two and a half inches. The structure is altered, becoming much more dense than natural. When examined microscopically, it shows no increase in the proper muscular tissue of the tongue, so that it is rather a case of false than of true hypertrophy. The increase of bulk is chiefly due to the production of a large quantity of highly vascular, nucleated connective tissue, which infiltrates and separates the muscular bundles. Beside this is seen a peculiar cavernous structure, composed of a number of intercommunicating spaces. These spaces contain a clear fluid, apparently lymph, and numerous spherical corpuscles. They appear accordingly to be dilatations of newly formed or pre-existing lymphatic vessels. We have seen this structure, which is described by Virchow and others with great clearness, in a case reported by Mr. Arnott.*

In other cases a true cavernous tissue containing blood, or something like a nævus, is formed. Sometimes there is said to be true muscular hypertrophy. The affection is sometimes congenital, and is said to be occasionally connected with cretinism or idiocy.

Atrophy of the tongue only occurs as the consequence of paralysis, from division of the hypoglossal nerve, or attacks of hemiplegia. It is of course confined to the affected side.

Changes in the Papillæ.—Dr. Salter gives an interesting account of the morbid changes which the lingual papillæ undergo. The circumvallate papillæ may be hypertrophied and form little tumours as large as peas. The epithelial caps of the conical or filiform papillæ may become extraordinarily elongated, so as to be half an inch long; they are of a dark colour, and look exactly like little brown hairs. Minor degrees of this condition are, we think, not uncommon. The papillæ sometimes becomes atrophied. "Mr. Lawrence mentions the case of a person in whom, from habitual drinking, the tongue was, for the greater part of its surface, destitute of papillæ: it was white, smooth, and opaque on the surface." Blood and lymph may be effused into the substance of the fungiform papillæ. The fur which so commonly collects on the surface of the tongue in disease, consists of detached, and more or less disintegrated epithelium, with varying proportions of amorphous matter. We can corroborate Dr. Salter's statement, that in some healthy persons the tongue is habitually furred. In very rare cases the frænum of the tongue is so short, that it is quite tied down to the floor of the buccal cavity, and cannot perform its proper movements. Minor degrees of the same condition are not infrequent, and gradually improve of themselves. In the opposite condition, "the movements of the tongue are too free; it can be inverted, and its apex thrown back into the pharynx, which embraces it," and thus the access of air to the lungs through the glottis is prevented. The sides of the tongue have been known to become closely adherent to the internal surface of the cheeks.

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* "Trans. Path. Soc.," 1872, vol. xxiii. p. 109.

Inflammation.—The tongue is liable to be affected by inflammation, or *glossitis*, as it is termed. This in some rare cases, said by Dr. Salter to occur most often in scrofulous persons, causes the formation of abscess. On the matter being evacuated, the tongue speedily returns to a healthy state. Sometimes a partial inflammation of the tongue is met with, the morbid process being confined to the portion of the base bounded in front by the V-shaped line of circumvallate papillæ. It occurs as an extension of tonsillitis, which we shall presently notice. Deglutition in these cases is seriously interfered with. The inflammation of the gums, which is produced by mercury, sometimes involves the tongue, and occasions in some cases very great and rapid swelling. It does not seem to have ever produced suppuration. One variety of glossitis has been distinguished by the term *erectile* by Dr. Salter. He describes "the morbid condition of the tongue in this disease as consisting in an enormous and rapid distension of the organ by blood, rendering it very large, hard. and stiff. The distension becomes so great that respiration through the mouth is quite prevented, and even can with difficulty be performed through the nostrils. Though the congestion becomes so intense that the organ is of a dark black colour, neither mortification nor abscess appears to have ever taken place. Free incisions give exit to the blood, and recovery ensues. Sometimes one-half of the tongue only is affected. In most cases it occurs in persons who are in perfect health, and without any manifest exciting cause.

Severe and deep *ulcerations* of the tongue may arise "from mere disorder of the alimentary canal," especially in debilitated persons. Some of these attended with much induration may bear a very close resemblance to cancerous ulcers. Constitutional syphilis produces small superficial circular ulcers, which sometimes extend in depth, and sometimes in length only. *Rhagades* or fissures result from the same cause; they often occupy the median line in the front part of the organ; they may be mere cracks, or extend three-quarters of an inch in depth, with irregular ulcerated edges. *Syphilitic tumours*, which are described by Dr. H. Salter under the name of *glossy tubercle*, appear to be of the same nature as the syphilitic tubercles termed *gummata*, found in various parts of the body. Ricord speaks of them as deep-seated tubercles of the submucous areolar tissue. They are composed of the usual fibronucleated tissue, and tend after a time to degeneration and atrophy. Often they cause very destructive ulceration.

Cancer.—The tongue is liable to be the seat of cancerous growths of the scirrhous and epithelial species. The former is described by Mr. Travers as at first being an irregular rugged knob, generally situated in the anterior third, and midway between the raphé and one edge. Ulceration sometimes takes place very rapidly; the surface at the same time throwing out luxuriant fungous growths: in other cases it "is very uneven, clear and bright granulations appearing in parts, and in others deep and sloughy hollows."

In an interesting case of epithelial cancer, carefully watched by Dr. H. Bennett, which may be taken as an instance of what is commonly seen, the first appearance of the disease was a small ulcer on the margin of the tongue This extended, in spite of its being shielded from the pressure of the teeth, and had hard everted edges, undermined some way by ulceration. These became more ragged, and here and there over the surface some degree of suppuration and sloughing occurred. Much improvement followed the excision of the tumour—the wound healed favourably. Not long after, however, the glands under the jaws enlarged, and were removed; and in about nine months after this, the disease returned in the tongue and proved fatal. The morbid growth which had been removed, presented on a transverse section a tract of white, indurated, infiltrated structure immediately below the ulcer, and above the muscular substance of the tongue. This indurated tract was half an inch thick posteriorly, and consisted of a fibroid structure enclosing débris of muscular fibre, and some of the characteristic circular loculi of epithelial cancer. The surface of the ulcer was covered with papillary elevations, which consisted chiefly of enlarged, softened, epithelial scales splitting into fibre, so as to form a kind of fringe, as in certain cells met with normally in deep layers of the epidermis.

This is the commonest form of cancer affecting the tongue. In its earliest stage it is stated to be a nodule below the surface, but it is not usually observed until the surface is broken, and the morbid growth appears as an ulcer, with raised edges and indurated base. The infiltration and ulceration spread rapidly, both superficially and in depth, till not only a great portion or even the whole of the tongue itself may be destroyed, but the destructive process may involve the gums, the fauces, and still more remote parts. Extension to the lymphatic glands takes place almost constantly, and the connection may sometimes be traced in the form of white cords running downwards from the base of the tongue, which doubtless represent lymphatic vessels enlarged and infiltrated with cancerous growth. The microscopic structure is that usually seen in flat-celled epithelioma, the great mass of the tumour being made up of epithelial scales, often arranged in loculi, and forming the well-known concentric or birds'-nest bodies. The same strictly epithelial growth may be found infiltrating the tissues beyond the apparent limits of the tumour itself, and is produced also as a secondary formation in the infected lymphatic glands. In this, as in other situations where epithelioma occurs, the epithelial formations in deep parts appear to be in direct continuity with the surface epithelium, which grows inwards, so as to infect the connective tissue and lymphatics.

Fatty tumours and *simple cysts* are occasionally met with in the tongue, and Dr. Salter mentions the occurrence of pedunculated *polypoid growths*, which seem to be of the nature of simple fibrous or fibro-cellular tumours.

MORBID CONDITIONS OF THE TONSILS.

Inflammation.—The tonsils are a common seat of inflammation and its consequences. In an acute attack they become swollen, sometimes enormously, so as to impede the respiration. The pillars of the fauces, and the soft palate, are also involved in the inflammation. Suppuration often occurs, and is, perhaps, the best result, next to complete resolution; but more frequently, the imperfectly subdued hyperæmia produces actual enlargement, and fresh attacks occurring, a chronic hypertrophy of the gland is the result. We have examined some enlarged tonsils which had been excised, and found their structure to be quite identical with that of the healthy gland, so that the alteration constituted a true hypertrophy.

Structure of the Tonsils.—It seems worth while to notice briefly this point, which we think is not well understood, as it explains in some measure the liability of these parts to hypertrophic enlargement. They are made up of a number of duplicatures and involutions of the mucous membrane, which, however, is differently constituted here to what it is in other parts in the vicinity. A vertical section shows the thin surface-layer of scaly epithelium with a thick underlying stratum, consisting of nuclei, or very slightly developed cells of a lymphatic type, which are imbedded in a stroma or network of fine threads, so that the whole constitutes what is now called "adenoid," lymphoid, or cytogenous connective tissue (*see* Part I., p. 153), such as is found under the epithelial layer in several parts of the intestinal tract. This layer is traversed by vessels, which are of capacious size in hypertrophied specimens, running up to the basement membrane which supports the layer of scaly epithelium. When there is any habitual hyperæmia, and consequent exudation, this submucous tissue readily gives origin to a new formation of similar substance, and so the enlargement continually goes on. The morbid condition which most resembles it is enlargement of the Peyerian patches, which we shall presently describe. This chronic hypertrophy may occur alone or else associated with similar enlargement of homologous tissues in other parts of the body, as the spleen and the lymphatic glands.*

Induration not unfrequently occurs as the result of inflammation, and depends, beyond doubt, on a fibroid development of the exudation. Rokitansky says, "In scrofulous subjects the tonsils are often affected, in addition to hypertrophy and habitual hyperæmia, with a peculiar blennorrhœa, and the purulent secretion not unfrequently becomes inspissated, so as to form tubercular cheesy plugs, or even chalky concretions. These, in their turn, keep up a perpetual state of irritation in the tonsils." Cancerous disease is very rare in this situation, but common indurated enlargement has often been spoken of as scirrhus. Tubercle is extremely rare in the tonsils.

II.—MORBID CONDITIONS OF THE TEETH.

The brief summary that we shall give of these conditions is taken from the excellent work of Mr. Tomes on the subject, to which we must refer for fuller information. *Malposition* of the other teeth is scarce more than a disfigurement, but when the wisdom teeth take a wrong direction the effects produced are sometimes very mischievous. Those of the lower jaw cause more serious evil by their wanderings than those of the upper. Sometimes the tooth, though not deviating from its proper position, is held down by indurated gum. Esquirol mentions a case in which mental derangement depended on this cause. The wisdom tooth may take a false direction inward or outward, and cause, by its pressure, ulceration of the tongue or the cheeks. It may grow directly forwards against the posterior surface of the second molar, which has proved the source of severe pain, resisting all treatment but that of extracting the offending tooth. Lastly, the tooth may advance against the coronoid process, causing disease and necrosis of the bone, and inflammation and abscess in the surrounding parts.

The teeth are very liable to *caries*, which is an affection very much of the same kind as that occurring in bones. Mr. Tomes believes that "the dentine, from abnormal (nutritive) action, loses its vitality," and therewith becomes liable to be decomposed by the fluids of the mouth. It seems necessary that both conditions should exist, that the tissue should be dead, and that the oral fluids should be in an acid state, capable of dissolving it. Test paper applied to carious teeth almost invariably shows the presence of free acid. Healthy saliva is alkaline, while that of dyspeptic persons is prone to be acid, and it is in such that caries is most apt to occur. The enamel is, of course, first affected, but a very small perforation through this tissue may exist with a considerable amount of disease in the subjacent dentine. It appears that when the acid solvent has once penetrated to the surface of the dentine, it extends laterally under the enamel, destroying, extensively perhaps, the body of the tooth, and undermining and eroding the enamel on its attached surface. The destructive process does not go on nearly so fast in the fang, which seems to possess a higher degree of vitality than the crown. A most interesting observation of Mr. Tomes demonstrates completely the vital nature of the actions going on in the dentine under the influence of disease. He shows that when a portion of dentine has become dead it is circumscribed by the consolidation of the adjacent living tissue. "The tubes become filled up, they are rendered solid, and the circulation is cut off from the dead mass by the obliteration of the tubes." It is remarkable, "that the consolidation does not go on gradually from without inwards, keeping in advance of the decay, but occurs at intervals." It is formed also in successive lines, a second one being produced when the first begins to be attacked, and afterwards a third, when the second gives way. The consolidated zones vary in width and in completeness, probably according to the vigour of the conservative action. Another interesting exhibition of vital action is displayed in the production of secondary dentine by the surface of the pulp, under the excitement of caries in the contiguous tissue. This vascular papilla, originally the formative organ of the dentine, which had for years confined its action to nourishing the perfected structure, under the stimulus of disease renews its formative action, and throws out a barrier between itself and the advancing mischief. How very analogous is this to the throwing out of lymph on the outer surface of a hollow viscus which is threatened with ulcerative perforation!

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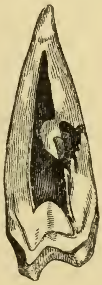
* See a case (described as "cancer") by Dr. Moxon: "Trans. Path. Soc.," 1869, vol. xx. p. 369.

The structure of secondary dentine is not so perfect as the original, and it is commonly vascular. Under the microscope "a transverse section of carious dentine, rendered soft, like cartilage, from the loss of its lime, presents a cribriform appearance. The tubuli are much enlarged and irregular in outline," differing entirely from their normal shape. This indicates that the solvent enters the tubes, and dissolves, first, their walls, and afterwards the intertubular material. In the consolidated zones the deposit obliterating the tubes is first removed, and afterwards their walls and the intervening tissue. A confervoid growth is very often seen on carious teeth, and on the tartar that may encrust them. Imperfect formation of the enamel is a frequent cause of caries.

It is mostly deficient on the surface, presenting deep pits with the intervening structure well developed; sometimes, however, small cavities exist in its substance, while the rest is perfect. Deep narrow fissures are often met with extending from the free surface to within the $\frac{1}{100}$th of an inch of the dentine; the enamel forming the walls of these is in parts perfect, in parts imperfect. Not only may the enamel be deficient, but it may be also imperfectly formed in various parts. The columns of its pulp consist of cells and granules, which, normally, become lost and fused in the homogeneous fibre; sometimes this fusion does not take place, and the granules remain, giving the enamel fibre a permanently granular aspect; or the cells do not undergo their wonted arrangement and elongation, and thus, though they calcify, do not form fibres. Sometimes the fibres of the enamel are not perfectly united at their margins; the resulting interspace may either appear as a broad line, or as a series of minute cells.

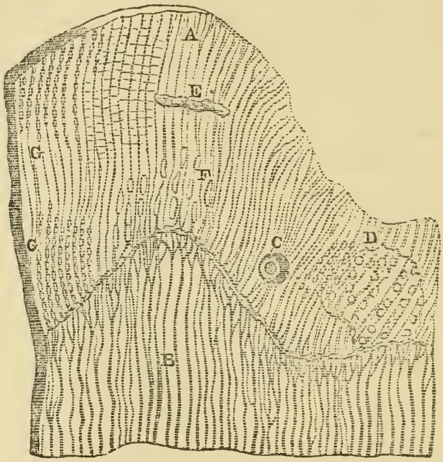
FIG. 125.



Drawing of tooth attacked by caries, with barrier of secondary dentine.

The teeth are subject not only to decay, but to death, to *necrosis*, which may be either complete or partial. After this has occurred certain physical changes commence. "The tooth gradually assumes a darker hue than natural, which increases in intensity till it is almost black. The dental periosteum gradually detaches itself from the fang, the tooth becomes loose, and unless held in by the crooked form of the roots, drops out. The surface of the fangs is generally rough, and, near the neck, dotted over with nodules of hard cream-coloured tartar, while the ends of the roots often look worm-eaten, as though absorption had commenced." Mr. Tomes compares the process of necrosis of a tooth with that which occasions the shedding of the antlers of a deer. In both the minute tubes or cavities through which nutrient fluid is conveyed, become obliterated by calcareous deposition; the whole tissue being consolidated into an inorganic mass. The dead tooth acts as a foreign body, causing inflammation and suppuration of the dental periosteum, as well as absorption of the latter, the alveolus, and gums. More serious effects are occasionally produced, "the periosteum of the alveoli becomes inflamed, together with the neighbouring parts:" and, if the case be still neglected, the adjoining teeth are not unfrequently lost, and necrosis of a considerable part of the jaw may also result. In some instances where the death of the tooth has taken place gradually patches of newly-formed cementum, thrown out by the irritated dental periosteum, adhere closely to the latter, and thus the tooth is held in its place. A single spot of necrosis in the fangs may cause inflammation and abscess and such severe pain, that the tooth, though otherwise quite healthy, is obliged to be removed. "Instances are not uncommon when the pulp of the tooth has died while the external surface of the fang has preserved its vitality. In these cases the dentine becomes discoloured, and gives a general dark appearance to the tooth. One of the three fangs of a molar tooth may alone be affected by necrosis, or the disease may be confined to one side of a single fang, producing absorption of the gum and alveolus on that side."

Fig. 126.



Imperfect formation of Enamel. (From Mr. Tomes' work.)

A. Enamel. B. Dentine. C. A perforation in the enamel. D. A cribriform layer of tissue in the enamel. E. A large cell lying transverse to the enamel fibres. F. Cells in the enamel about the apices of the coronal dentine. G. Lines of minute cells between the enamel fibres.

The layer of osseous tissue, called cementum, which coats the fangs of the teeth, is liable to become hypertrophied. This may proceed to such an extent that the fang near its extremity may be twice the diameter of the neck. It results from irritation of the dental periosteum, which may itself be occasioned by caries, or necrosis of the tooth. The enlarged fang necessarily compresses and irritates the nerves which pass through the orifice at its extremity to the pulp; and this irritation may be the cause of epileptic seizures, or paroxysms of neuralgic pain. The fangs of the teeth are occasionally absorbed to a greater or less extent, in some rare instances to the same extent that those of the temporary teeth are. The *dental pulp*, a highly sensitive and delicate structure, is very liable to be affected by severe pain from the irritation of caries in the tooth, or even from disease of the pulp of an adjacent tooth, or one situated on the opposite side or even in the other jaw. This is an excellent example of the reflection of sensations. Inflammation often attacks the dental pulp, changing its natural pinkish-grey colour to a bright scarlet, and terminating very commonly in its suppuration and death. Sometimes when there exists an opening, formed perhaps by caries, into the pulp cavity, the inflammation affects onlya part of the pulp. This is intelligible, from the circumstance that the secreted matter has a channel of exit, and does not diffuse itself over the rest of the pulp, and also because the irritation of the oral fluids and of the carious dentine is confined to the adjacent part of this structure. The inflammation, after having caused the destruction of the pulp, may extend to the dental periosteum and occasion the death of the fang; it may even extend further to the periosteum of the jaw, and propuce necrosis of the bone. The dental pulp may be removed by *absorption* after the cavity of the tooth is laid open by caries, or it may *ulcerate* or perish from *gangrene*. Or again, it may become the seat of a fungoid growth, not of cancerous nature, which is sometimes termed polypus.

The *dental periosteum* may be inflamed acutely or chronically. When the former takes place it causes the periosteum to separate to a greater or less extent from the surface of the fang, leaving a cavity which is occupied by pus. The cavity becomes enlarged at the expense of the alveolus, and this chiefly at the apex of the latter. Sometimes the pus makes its way into the mouth at the neck of the tooth, having detached the periosteum up to that point, but more commonly the abscess advances through an opening in the alveolus into the gum, whence it ultimately makes its way to the surface. In unhealthy states of system the disease may involve the bone adjacent, and cause necrosis to a considerable extent; or it may creep on and affect the periosteum of contiguous teeth. The inflammation rarely arises spontaneously; most often it is the sequel of inflammation of the pulp. In the chronic form there is no tendency to the formation of abscess, but there may be a slight discharge of pus from the edge of the gum. "The tooth becomes loose, the alveolus absorbed, and the edge of the gum inflamed. The gum gradually sinks with the absorption of the alveolus, and the tooth drops out or is removed." Sometimes partial chronic inflammation occurs, causing "the periosteum about the extremity of the tooth to become thickened and nodulated." The *alveoli* are liable to necrosis from various causes as other bone is; they undergo absorption in old age naturally, and sometimes prematurely in persons who have been subjected to long-continued salivation, or whose gums have been rendered unnaturally vascular by other causes; and lastly, they are sometimes the seat of exostosis, which gradually, as it increases, extrudes the tooth.

III.—ABNORMAL CONDITIONS OF THE PHARYNX AND ŒSOPHAGUS.

Congenital Malformations.—This part of the alimentary canal may be congenitally absent, or may terminate in a cæcal pouch, or be fused with the trachea, or be dilated into a sac, or in rare instances be traversed by separate fissures. The latter condition has been called fistula colli congenita, and has been attributed to imperfect closure of the third or fourth visceral cleft. The fistula opens externally, at the inner border of the sterno-mastoid muscle.

Dilatation.—The pharynx and œsophagus sometimes become dilated throughout, their parietes, and especially the muscular tunic, being hypertrophied. Rokitansky alludes to one case in which the passage was large enough to admit a man's arm. In some instances of a less degree of dilatation, the coats are relaxed and attenuated. This is sometimes a consequence of obstruction at the cardiac orifice of the stomach, but sometimes occurs without any obstruction, apparently from paralysis of the muscular walls, produced by chronic inflammation. Partial dilatation appears in the pouches, which sometimes are formed by all the coats of the canal, sometimes consist of the mucous membrane only. In the latter case, "the mucous membrane is protruded between the muscular fibres, and becomes dilated by the food that enters; it is at last forced out in the shape of a cylindrical appendix, which lies between the vertebral column and the œsophagus, in a line with the axis of the pharynx, so that all ingesta pass into it, and death from starvation results." This change is chiefly seen at the lower part of the pharynx, but similar dilatations are formed lower down in the œsophagus, and are produced, according to Rokitansky, by the contraction of bronchial glands, which have formed adhesions to the wall of the œsophagus and draw a portion of it outwards. The most remarkable cases of dilatation of the œsophagus are, however, those in which no obstruction or other cause can be found.

The œsophagus is liable to be *constricted*, either by the compression of external growths or by cicatrices in its own walls, the results of former ulceration or sloughing, or by cancerous formations in its coats. A very obstinate form is the consequence of the action of irritant or caustic substances.

Inflammation.—Acute inflammation occasionally attacks the pharynx, or rather its mucous lining, chiefly by extension of the disease in Cynanche tonsillaris, or simultaneously with the fauces in Scarlatina anginosa. Sometimes the affection, though then generally less acute, is independent, and constitutes Cynanche pharyngea. Inflammation of the submucous connective tissue or cellulitis, occurs most often in children, and goes on to suppuration, forming the retropharyngeal abscess. It is sometimes connected with caries of the vertebræ, and does not then, strictly speaking, belong to the pharynx. Chronic pharyngitis is very common, especially in persons of an atonic habit, or who speak much from the throat. The mucous membrane appears slightly swollen, and with an uneven granular surface, coloured by an uniform redness, and denuded to some extent of its investing epithelium. In many cases the redness is more patchy, and seems to affect more the small veins and adjacent capillaries, and is of a darker tint. M. Chomel has described one form of this affection, in which the mucous follicles are specially the seat of morbid action. He says the arch of the palate is seen to be covered by small red points, which are more thickly disseminated on and near the uvula. These become more numerous and larger as the disease advances, till at length they run into each other, forming ridges and raised patches, between which only a small part of the mucous membrane retains its natural appearance. It occurs much more frequently in males than females, and chiefly between the fifth and seventeenth years.

Croupy inflammation sometimes extends to the pharynx and commencement of the œsophagus, and, on the Continent at least, diphtheritic exudations are by no means infrequent in this situation. They are essentially similar to those which form on the buccal mucous surface, which have been already described. The pustules of variola are occasionally met with in the pharynx, and those which are caused by large doses of tartar emetic, in the lower third of the œsophagus.

Fibrous tumours originating in the submucous or deeper-seated areolar tissues may grow inward, and obtaining an investment of mucous membrane hang down into the œsophagus in the form of a polypus. Sometimes they remain without thus protruding, in the submucous tissue. Tuberculous disease is very rarely found in the pharynx or œsophagus. *Cancer* is more frequent in the œsophagus than in the pharynx, in the proportion of thirteen to four; in the former it mostly affects the upper part just below the larynx; in both parts it almost always assumes the form of infiltrated scirrhus, from an ulcerated basis of which soft fungoid growths may afterwards sprout. In the pharynx, according to Dr. Walshe, it generally presents, at least in the early period, "a hard imperfectly circumscribed mass," which may form a tumour visible externally. In the œsophagus it mostly constitutes an annular layer, constricting the canal for a variable distance, and often by extending outward producing adhesion of the diseased mass to the spinal column. We have observed one instance in which encephaloid cancer in this situation had proceeded to a considerable extent without producing any symptoms. In the œsophagus of a man who died with peritonitis there was a thick mass of encephaloid, ulcerated on the surface surrounding the lower part of the canal just above the cardiac orifice of the stomach for an inch and a half. It had evidently originated in the submucous tissue, and grown inward, not contaminating the other coats. Higher up there were several smaller submucous tumours. Rokitansky mentions the formation of ulcerated openings communicating with the trachea and the bronchi, or even with the aorta and the right pulmonary artery.

Epithelioma of the pharynx hardly occurs except in connection with the same disease in the mouth, and propagated from there.

In the œsophagus, on the other hand, it is comparatively frequent, and according to the unanimous testimony of recent observers much more frequent than encephaloid or scirrhus, with ulcerated forms of which it has doubtless been confounded. According to Dr. Wilks, it is most often found opposite to the bifurcation of the trachea, where it forms a long cancerous ulcer, some three or four inches in length, with raised edges, the surrounding mucous surface being sound. Sometimes it involves the whole circumference of the tube, but if not, then the disease is always more advanced on the anterior wall. The disease is almost always quite local, affecting only the parts immediately adjacent and the neighbouring lymphatic glands, but we have seen secondary growths in the liver in some cases.

Round Ulcers.—Simple round ulcers of the œsophagus resembling the round ulcers of the stomach, and like these, owing their form and other peculiarities to the action of the gastric fluid, are met with, though rarely, at the lower end of the tube.

Post-mortem Softening.—The lower part of the œsophagus is liable to softening from the action of the gastric fluid after death, which attacks especially the left side, which adjoins the left pleura, and occasions perforation and effusion of the contents of the stomach into this serous cavity. It is often associated with similar softening of the stomach, but may occur independently.

IV.—ABNORMAL CONDITIONS OF THE PERITONEUM.

Congenital deficiencies in the peritoneum will, of course, exist when any of the viscera which it invests are absent, or imperfect, or when the walls of the abdominal cavity are in a like state. It appears also that the various folds may of themselves be imperfectly developed, *e.g.*, the omentum or mesentery may be unnaturally small or absent. On the other hand, these same folds may be of unusual dimensions, as when a mesentery of more than common length allows the intestines to float up to the surface of an abdomen distended by ascites. Certain pouches, which may by enclosing and incarcerating portions of intestine produce hernia, like those of the external diverticula of the peritoneum, have been described especially by Treitz. He distinguishes the fossa duodeno-jejunalis, formed by the side of the aorta, near the origin of the inferior mesenteric artery by a fold of peritoneum surrounding the duodeno-jejunal flexure; the fossa intersigmoidea formed on the under surface of the mesocolon of the sigmoid flexure; and the fossa subcæcalis formed by a separation of the layers of the mesocolon belonging to the ascending colon, and thus lying behind the cæcum.

The first-mentioned condition is the commonest of the three; it has been in some cases the cause of hernia and fatal obstruction.

We have seen an instance of the last in which three such pouches were formed one above the other between the mesocolon and the abdominal wall, but had given rise to no symptoms though they contained coils of ileum.*

Peritonitis.—The peritoneum is extremely liable to *inflammation* which may be of various degrees of acuteness, or may be chronic *ab initio.* The former is extremely common, and results not only from all causes of irritation applied to it, but also originates spontaneously; or, as Rokitansky avers, in consequence of the rheumatic poison. The inflammation varies much in extent, very often it is general, affecting the whole membrane, but often also it is partial, confining itself to a certain region. The simplest instance of partial peritonitis which we can take is, perhaps, that which occurs when an ulcer is making its way through the walls of the intestine, and threatening to perforate them. Opposite the threatened spot a patch of injected vessels appears on the serous membrane, which pour out a fibrinous exudation forming a protecting investment, or an adherent medium uniting it to adjacent parts. Inflammation, however, when set up at one part is very prone, as in all serous membranes, to propagate itself to the surrounding; and thus it very commonly happens that peritonitis, which commences in one locality over an inflamed or irritated organ, diffuses itself over the whole membrane till it becomes general. In the early period injected vessels are very distinctly seen in the inflamed membrane, forming streaks or patches of redness. The injection, however, is seldom very strongly marked, except in the spaces formed by adjacent loops of bowel, which is, perhaps, due in part to the readiness with which exudation takes place. This is often seen as a delicate thin layer of fibrinous matter closely investing the inflamed surface, sometimes it is so scanty that it is scarcely discernible, unless the surface is carefully scraped, or adjacent intestinal convolutions are separated from each other, when it appears as minute filaments stretching across the interspace. The inflammatory lymph sometimes collects in the furrows between convolutions of intestines pressed together, and is more manifest there than elsewhere. In instances of severe inflammation the inflammatory products are often very abundant, and much puriform is mingled with the fibrinous matter. Serous fluid is also poured out often in considerable abundance, and is rendered turbid by flakes and molecules of fibrine and pus corpuscles diffused throughout it. In peritonitis attacking persons who are in an asthenic state the serous and puriform products generally predominate.

From the ease with which inflammation of the peritoneum can be set up, experimentally, it has been specially studied as an example of inflammation in general. Peritonitis set up by rupture of the intestine appears to resemble precisely that produced experimentally by injecting irritating substances; and may be taken as a type of acute inflammation. In such a case we find, beside hyperæmia, emigration of blood corpuscles, and production of fibrin, germinative changes, especially in the endothelial plates (Fig. 8, page 89). These appearances seem, however, to be similar to what may be normally seen in the peritoneum, differing from these chiefly in their greater intensity.

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* Treitz, "Hernia Retroperitonealis," Prag. 1857; Pye-Smith, "On Retroperitoneal Hernia," "Guy's Hospital Reports," vol. xv. ser. 3; Payne, "Trans. Path. Soc.," 1874, vol. xxv.

Adhesions.—Adhesions are very often found in the peritoneum, connecting the visceral and parietal layers together, and are sometimes of considerable length; in many instances, no doubt, they are the result of partial inflammations giving rise to fibrinous exudation, which is afterwards transformed into areolar tissue; in other cases we are inclined to think the growth takes place with little or no preceding hyperæmia, and appears to be effected by the same natural germinative process to which we have already alluded, and which is also the cause of the gradual thickening and shortening seen in the omentum with advancing age. The inflamed membrane becomes somewhat thickened by the effusion taking place in its own texture. Bands of adhesion sometimes become the cause of fatal incarceration of the intestine, an opening being formed by these means, into which a coil of intestines passes, and after a time having become distended, is strangulated by the abnormal band. A case of this kind is recorded in the report of the Pathological Society, 1851-52, in which seven or eight inches of the lowest part of the small intestine were strangulated by a ring formed by a strong fibrous band passing from the mesentery to the anterior surface of the rectum.

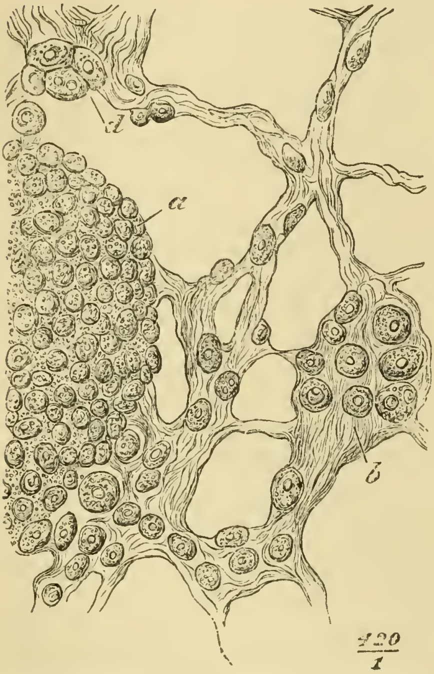
Suppuration, as has been said, is not unfrequently a result of acute peritonitis, the purulent matter being, as it were, smeared all over the surface of the membrane; sometimes, however, it takes place in a single part, and forms a circumscribed abscess. On opening the abdomen of a female who had been attacked with peritonitis after the operation of ovariotomy, and who survived several weeks after the inflammation had been subdued, there were found not only traces of lymph on the surface of the intestines, but a quantity of well-formed pus in the interior of a cavity formed by adjacent convolutions. Had life been prolonged the pus might have made its way by ulceration into the intestinal tube, and thus been evacuated. Rokitansky in mentioning this occurrence speaks of the abscess sometimes discharging itself through the abdominal parietes, or opening this way as well as into the intestine, so that a fistulous communication with the bowel is the result. Purulent discharges sometimes take place from the umbilicus. If not evacuated, pus sometimes becomes shut off or encysted in special portions of the peritoneum, such as between the liver and diaphragm, or in either inguinal region; and we have become convinced that in such cases absorption of the solid portions of the pus may take place, only serous fluid remaining in a thick-walled cyst lined with fibrinous flakes, as happens also in limited empyema or in softening of the brain.

The intestines are very commonly distended by gas in acute peritonitis, which has been thought to be secreted by the mucous membrane under the influence of irritation, but is possibly derived from decomposition of the intestinal contents (p. 72). At the same time there is reason to believe that the action of the muscular fibres is more or less interfered with, if they are not actually paralyzed, and hence the distension being unopposed is greater than it otherwise would be. Rokitansky says that "hæmorrhagic exudation is frequently seen on the peritoneum; it forms large, saturated coagula, disposed in thick layers."

Chronic peritonitis of a simple kind is not of common occurrence; a case is, however, recorded by Andral in which serum turbid with albuminous flocculi was found in the serous cavity after death, while ascitic effusion had existed for more than the last month of life, unattended with pain or any evident symptom of inflammatory action. We think we have seen a somewhat similar case. There is, however, another form of what may, perhaps, be termed chronic peritonitis, though we doubt very much its essential dependence on any inflammatory process. It is essentially partial, occurring only on the serous coverings of certain organs. In this the serous surface is invested closely for a greater or less extent by a firm, whitish, false membrane, which can be pretty easily detached from the subjacent peritoneum, and appears, when held up to the light, of much thinner texture in some spots than in others. The situation in which the false membrane is most completely formed is upon the surface of the liver, to which it forms sometimes a complete capsule, compressing and atrophying it, and giving rise to ascites from interference with the free passage of the blood through the structure of the gland.

Tubercular Peritonitis.—One of the best marked varieties of chronic peritonitis is that which is often justly called "tubercular," from its being essentially dependent on the presence of tubercles in the peritoneum. These appear sometimes as semi-transparent grey granulations, sometimes are more opaque, though still of miliary dimensions. They are diffused everywhere throughout the peritoneal surface, but are said by Dr. West to be most numerous on the surface of the diaphragm, or on the abdominal walls in the neighbourhood of the spleen, while the parietal peritoneum is not so much affected as other parts. The chief tubercular product is sometimes in the omentum, and may assume the form of yellow caseous masses. Inflammatory irritation is produced by the tubercles acting as foreign bodies, exudation of lymph takes place, and adhesions are formed between adjacent parts of the serous surface which are often so close and dense that the intestines or other viscera are torn in making an attempt to separate them from each other. We have seen the whole of the serous sac in this way entirely obliterated. There is usually some serum in the peritoneal cavity, but no puriform matter, unless, as occasionally happens, acute inflammation has supervened upon the chronic and proved fatal. The tuberculous material sometimes undergoes softening, as in other situations, and the extension of this process may cause perforation of the walls of the intestines, and either establish unnatural communications between distant parts which have become adherent together, or lead to the effusion of the intestinal contents in the serous cavity. The latter event, however, is more likely to be produced by the softening of tubercle in the submucous tissue, since it is often simultaneously present there as well as in the mesenteric glands. There is no relation apparently between the amount of deposit in the peritoneum, and in these two other localities. The lungs and bronchial glands are often tuberculous when the peritoneum is affected, but often in a much less degree, and they may sometimes be altogether exempt. Rokitansky describes the muscular tunic of the intestines as being still more affected in this disease than in acute peritonitis; it becomes pale, is easily lacerated and broken up. This gives a further reason why laceration often occurs on attempting to separate the intestines which are matted together.

FIG. 127.



Miliary tubercle of the omentum, and changes in surrounding parts.

a, Tubercle completely formed.
b, Cluster of small cells forming an incipient tubercle.
d, Proliferation of endothelium.

From an original preparation ("Trans. Path. Soc." vol. xxi. p. 198, pl. v.).

The acute form of this complaint is usually, if not invariably, either subsequent to some chronic form of tubercular or degenerative inflammatory disease elsewhere, or a part of general acute tuberculosis. The chief features of the disease are the production of grey miliary tubercles in large numbers; and should its course be somewhat more prolonged, of fibrous adhesions between adjacent surfaces. The masses of grey tubercles may attain a very considerable thickness without undergoing any caseous transformation. In fact, it may be stated generally, that tubercle in the peritoneum has a tendency to fibrous rather than to caseous change.

Condition of Peritoneum in Ascites.—The peritoneum often appears somewhat thickened, of a dull, dense, whitish sodden aspect in cases of chronic ascites. We have examined the membrane thus altered, but were unable to find any very marked alteration in texture, only that the tissue seemed more granular and less purely fibrous than natural. A local change of somewhat the same kind is often seen in the peritoneal covering of the liver and the spleen. In these it forms dense whitish patches, which shade off gradually at their margins, and are for the most part quite free from bands of adhesion on the surface. The change in the splenic capsule is often so great that it has been termed "cartilaginification." When the exudation, which in all these cases takes place into the subserous tissue, contracts and draws the part together, it produces "lobulated laminæ, and projecting granulations," of a firm, dense structure. Calcareous matter is sometimes deposited in these fibrous formations, and gives rise to "compact, smooth, or uneven lobulated plates of varying thickness." It appears from Rokitansky's account, that false membranes, the result of inflammation, may be so disposed as to form serous cysts, which obtain an internal smooth lining, and are either pedunculated or sessile on the peritoneum.

Cancer, or some form of malignant growth, attacks the peritoneum in some very rare cases primarily, but most often by an adjoining growth extending to it, "perforating it, and penetrating into its cavity." The disease is sometimes of the Encephaloid variety, but more often of the Colloid. The latter, when the process of development is acute, is often spread over the entire serous surface, in the form of small, miliary nodules, in some parts clustered together. "Sometimes it occurs as a layer of areolar cancerous tissue, varying in thickness, or as a circumscribed, round, lobulated aggregation. The omentum is very commonly found to shrivel up and to degenerate into a transverse band; or, in the opposite case, with an enormous increase of size into areolar cancer." This generalized cancer of the peritoneum may occur either in consequence of the bursting of a softened tumour in some adjacent organ, when the small growths appear as if sown over the surface; or else by extension without any actual softening or distribution of material; in which case the peritoneal growth is usually more local. We have seen a cystic sarcoma cause by rupture into the peritoneal sac the growth of hundreds of small tumours on its suface. Sarcomatous growths not uncommonly originate in the *post-peritoneal cellular tissue* just in front of the spine. In this part they are firmly adherent to the vertebræ; are of homogeneous (probably firm scirrhous) texture, and confounded with the crura of the diaphragm. In its peripheral parts the growth has a more loose and lobulated structure. Dr. Walshe, tracing the progress of the mass from its point of origin, says, "it spreads upwards, extends to the stomach, presses under the liver, penetrates between the laminæ of the transverse mesocolon, twists round the duodenum and pancreas, and, pushing forward the stomach (with the small curvature of which it contracts adhesion), forms a tumour in the epigastrium. Such tumours mould themselves upon these various parts and organs in so close a manner, that, after separation, the surface of the mass retains the impressions of the adjoining viscera." The course of these growths, though rapid, is often for a long period unattended with pain or disturbance of the system. Primary malignant tumours of the peritoneum are generally, if not always, sarcoma.

V.—ABNORMAL CONDITIONS OF THE STOMACH.

Congenital Abnormalities.—In very imperfect monstrosities, especially the acephalous, the stomach is either wanting, or very imperfectly developed. It is also absent occasionally, according to Rokitansky, in individuals otherwise normally built, and provided with a well-developed intestinal tube; or it may be only indicated by a small saccular dilatation of the œsophagus.

Variations in Shape and Size.—The *shape* of the stomach is sometimes found remarkably altered; its cavity being partially divided into a cardiac and pyloric portion by an annular contraction, or even still further subdivided into three or four sacculi, so as to present some resemblance to the multiple stomachs of ruminants. These peculiarities of shape may either result from congenital malformation, or at least in their minor degrees, from irregular contractions of the muscular coat, or from destruction of substance and subsequent cicatrization. Sometimes the stomach deviates from its usual shape, in the way of assuming greater simplicity; it is destitute of its cardiac cul-de-sac, and the œsophageal opening is quite at its left extremity.

The stomach is liable to great variations of *size;* these within certain limits are physiological, and are manifestly contemplated in the peculiar convoluted disposition of its mucous lining. A healthy stomach, when empty, naturally contracts upon itself, and this to such an extent that its cavity is well nigh obliterated. This is purely the effect of the unopposed action of its muscular coat, and is no evidence of disease in the viscus. It may proceed from starvation, or from stricture of the œsophagus. Contraction of the cavity of the stomach in a less degree may be produced by hypertrophy, and cancerous disease of the coats, or by the cicatrization of extensive ulcers. Partial or "hour-glass" contraction is attributed to tight-lacing in women. *Dilatation* of the stomach, often to a considerable extent, is of very common occurrence, and depends partly on copious production of gas, and partly on loss of contractile power in its muscular fibres. In its more extreme degrees, it is generally the result of obstructive disease at the pyloric outlet, in consequence of which the ingesta accumulate within the cavity.

The distension in such cases is sometimes so enormous that the stomach extends over the entire abdominal cavity, the greater curvature reaching the pubes. Rokitansky says, "that repeated repletion, in consequence of a morbid appetite," may become the cause of as great distension as when the pylorus is obstructed; or that this may also "occur as a result of paralysis from concussion, traction, or dislocation, produced by large scrotal herniæ, and that it kills slowly with vomiting, with or without gangrene of the mucous membrane, under symptoms of complete paralysis." The dilated stomach may show muscular hypertrophy in chronic cases, or in rapid cases, atrophy.

Changes in the Coats.—The coats of the stomach may be abnormally thick, either in consequence of cancerous disease, or from simple hypertrophy of the muscular layer. The pyloric outlet is generally the part where muscular hypertrophy shows itself, and here it seems to affect more especially the layer of annular fibres. Thickening may also be a consequence of chronic inflammation. *Atrophy* of the coats, speaking generally, is most commonly observed in cases attended with much emaciation; it occurs also sometimes in consequence of extreme dilatation of the cavity, sometimes spontaneously. The muscular layer is, in most cases, the one most palpably affected; the mucous membrane is, however, not uncommonly atrophied also, as we shall more particularly describe when speaking of textural changes.

Abnormal Situations.—The following list of abnormal situations, which the stomach may occupy, is given by Rokitansky: "It may lie external to the abdominal cavity in eventration, and in umbilical hernia; in the left side of the thorax, the diaphragm being wholly or partially absent on that side;" it may lie vertically, as in the fœtal state; or with the fundus on the right side, as in general lateral transposition. The foregoing are congenital malpositions; the following acquired. The stomach may protrude externally after extensive wounds, or make its way into the thorax, after injuries to, or ruptures of, the diaphragm; it may be carried down into large hernial sacs, especially umbilical and scrotal; or be displaced by new growths or enlargement of adjacent organs; or sink lower itself, in consequence of increase in size, as in the case of a scirrhous pylorus.

INFLAMMATION OF THE STOMACH.

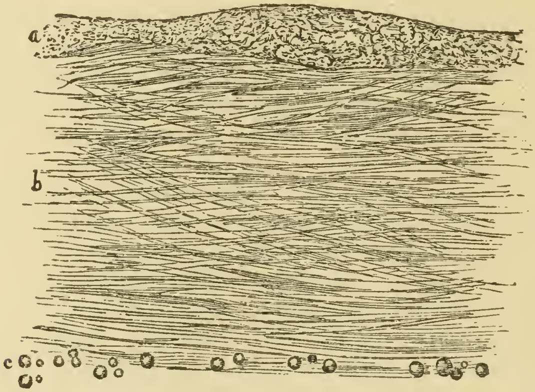
Acute inflammation of the stomach is characterized by intense redness of the mucous surface from injection of the vessels and swelling. The redness is either diffused or in the form of dendritic ramifications, and, in addition, numerous minute ecchymoses are often met with. The swelling depends upon enlargement of the peptic glands and some interstitial effusion. The surface is covered with thick and sometimes blood-stained mucus. Occasionally superficial erosions or commencing ulcers are seen. The submucous tissue is also injected; the muscular coat usually relaxed, so that dilatation results. These changes seldom extend over the whole surface, but are usually confined to one part, mostly the pyloric half. In arsenical poisoning, however, the arsenic being applied to a wound in the skin, the hyperæmia, which is very intense, chiefly affects the splenic region, and, what is curious, the upper parts of the folds are much more congested than the lower.

Acute inflammation of the stomach but rarely, if ever, occurs, except as the result of irritants directly applied to it. The cases related by Andral give proof, however, of its existence as an idiopathic affection, or as the sequel of rheumatism, or of epidemic cholera. We quote the account he gives of the morbid changes. In case 1, the stomach was strongly contracted, so as to be nearly the size of the transverse colon: "Its inner surface, over nearly its entire extent, was of a brownish red. This colour had its exclusive seat in the mucous membrane, which had become in every part very thick, and was at the same time very friable. On its free surface there were discovered a multitude of small red or blackish points, which seemed to have their principal seat in the villi; however, beneath these the body of the mucous membrane was red, and, as it were, penetrated with blood; in no part could this membrane be detached, it gave way under the forceps, and in several points it resembled a pulp without any consistence." Near the pylorus, the mucous membrane resumed its normal consistence, and was of a greyish colour. In case 2, also, the stomach was strongly contracted. Its mucous membrane was of a dark red colour, over the entire surface of the great cul-de-sac, and over all the posterior surface from the cardia to the pylorus. This redness penetrated the entire substance of the membrane, which had lost its consistence in every part where it was red; in some points it was merely a sort of pulp. Towards the anterior surface the mucous membrane presented a slate-coloured tint, without its consistence being much changed: near the pylorus some mammillation was observed. The surface was covered by a viscid thready mucus. In another case, also idiopathic, the parietes of the stomach were remarkably softened: "Over all the left portion of this viscus, its tunics, from the peritoneal to the mucous, had no longer any consistence; they gave way under the fingers as a sort of pulp. Wherever this softening existed, the parietes of the stomach were of a dark red colour, and as it were ecchymosed. Near the pylorus the parietes of the stomach resumed their natural consistence," and were of a greyish tint. In persons dying of gastritis, consecutive to a malignant cholera, Andral found, at an early period of the disease, the mucous membrane red and softened; at a later period it was sometimes in the same condition, sometimes brown or slate-coloured, and its tissue thickened and indurated.

In an excellent instance of intense hyperæmia of the stomach, which we had the opportunity of examining, the whole mucous surface was of a deep red, almost black, the subjacent tissues were much less affected. The cavity was empty, and the organ was much contracted. The surface was uncovered by mucus, only a little alkaline fluid lay in the furrows between the rugæ. The capillaries were gorged with blood in every part of the membrane, they were seen running parallel to the tubes in their whole length, but those which adjoined the free surface (which are always the most congested) had given way in numerous spots, and saturated the tissue round them with extravasated blood. The tubes were healthy, and there was no apparent exudation among them. The patient died with cardiac hypertrophy and general dropsy. The liver and spleen were much congested, and it is pretty certain that the hyperæmia of the stomach was passive rather than active.

Catarrhal Inflammation.—As Rokitansky remarks, we have rarely, if ever, the opportunity of observing the first stage of acute catarrhal inflammation of the stomach, but we may reasonably infer that it consists, as in other parts, of a more or less considerable hyperæmia, accompanied by a copious exudation of mucus upon the surface, with little or no fibrinous exudation in the substance of the tissue. A chronic catarrhal state is by no means uncommonly met with, the anatomical characters of which Rokitansky enumerates "as a dark reddish-brown, or slate-grey, or even blackish-blue discoloration of the mucous membrane, copious secretion of a stone-coloured, occasionally glassy pituita, thickening, increased condensation, and induration, *i.e.*, hypertrophy of the mucous membrane, which presents itself in various degrees: (α) In the lowest degree, the mucous membrane shows simply an increase of thickness and hardness in its tissue; (β) in a higher degree it presents, in addition to its increased thickness, an uneven, racemose, or warty surface, a surface mamellonée; (γ) in a still more advanced degree, it forms prolongations in the shape of permanent firm folds or of polypus. The submucuous cellular tissue, and the muscular coat, also participate in this hypertrophy in various degrees—the entire parietes of the stomach presenting unusual thickness, firmness, and hardness. The pyloric portion is the chief seat of chronic catarrh, and it is there that hypertrophy of the mucous and other membranes is most prominent." Andral remarks, that in chronic gastritis the mucous membrane may appear after death to be in a perfectly natural state, or at least to have undergone no alteration discernible by the eye. The subjacent tissues, and particularly the submucous areolar tissue, are, however, in these cases more or less affected. In the majority of instances, however, the colour, the consistence, and the substance of the membrane are variously changed. A grey slate, brown, and more or less deep black, tint are often observed, as well as sometimes a dull white, milky aspect. In regard of consistence, the mucous membrane may be indurated or softened; the latter is more frequent than the former. Induration may exist with all the different shades of colour just mentioned. With respect to its substance the mucous membrane may be thickened or attenuated, or may remain unaltered. A state of thickening may coexist with induration or with softening; the former combination is peculiar to chronic gastritis, and affords a good example of false hypertrophy. Induration, like the thickening, may be partial or general. Attenuation of the gastric mucous membrane is most often met with towards the great cul-de-sac, in the same situation where softening is most frequent. Andral says, "Sometimes, however, I have found the mucous membrane towards the pylorus so attenuated that it resembled a sort of transparent, extremely fine web. On attempting to raise it, it was changed into a reddish white pulp, as happens in certain degrees of softening." He admits, what we shall presently remark, that this attenuation may occur as a pure atrophy totally independent of inflammation.

Fig. 128.

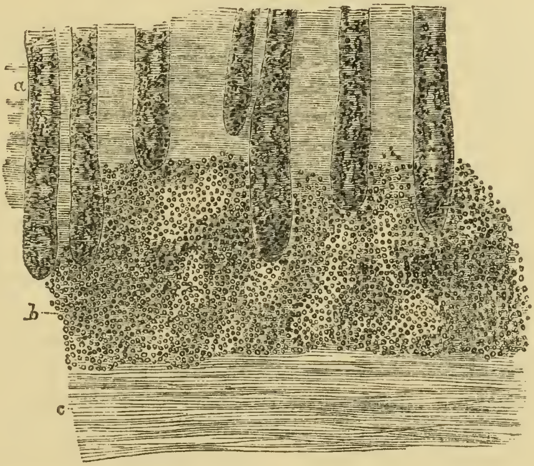


Vertical section of mucous membrane of stomach, the tubes being completely wasted and replaced by fibroid tissue.

a, Remains of mucous membrane. *b*, Fibroid tissue. *c*, Fat cells.

Minute Changes in the Stomach.—We have examined carefully with the microscope more than a hundred stomachs taken indifferently, and have published in the "Assoc. Journal" for Oct. 7th and Jan. 27th, 1854, the details of the following varieties of change:—(1) One of the commonest, especially in its minor degrees, consists in the infiltration of a low fibroid tissue loaded with nuclei or small cells among the tubes, which themselves undergo atrophy, so that at last the mucous membrane totally loses its tubular aspect, and becomes a mere fibroid stratum, more or less densely set with nuclei throughout. In this state the basement membrane may still persist, and the thickness of the tissue be little diminished. In some instances the nuclei disappear, and the fibroid stratum develops fibres more decidedly. (2) There are formed masses of nuclei, or small cells, most often at the bases of the tubes encroaching upon them, often also in the substance of the mucous membrane, and sometimes at its surface:—these are sometimes circumscribed, sometimes diffused, and then pass into the preceding forms by gradual shades. (3) The nuclear deposits sometimes seem to give rise to cystic cavities, or these may form from dilatations of the tubes, or arise, *de novo*, as in other situations. (4) The mammillated condition appears to depend on a process of local atrophy, at least in most cases, the tubes being wasted in the track of the furrows, which sometimes are so deep as to fissure the membrane down to its

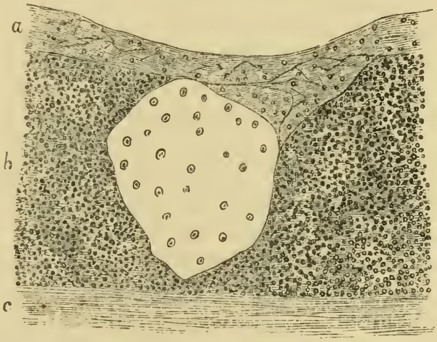
FIG. 129.



Vertical section of mucous membrane of stomach, showing the lower parts of the tubes, and a nuclear mass extending among them upwards.

a, Tubes. *b*, Nuclear infiltration. *c*, Submucous tissue.

FIG. 130.



Cavity formed in the mucous membrane of stomach, by the disintegration of a nuclear mass. The surrounding tissue is pervaded by nuclear deposit.

a, Basement membrane of surface.
b, Altered mucous membrane.
c, Submucous tissue.

corium. (5) A fatty state is very commonly met with, and in two forms, one in which the epithelium is bulky and the tissue healthy, or nearly so; the other, where the epithelium is atrophied, in consequence, generally, of pressure by new-formed fibroid tissue upon the tubes. (6) The tubes in the pyloric region are often found changed in the following manner: the continuous row of tubes is interrupted, and there are seen at intervals instead groups of convolutions containing a fatty wasted epithelium, and not possessing any manifest outlet on the surface. We think that inflammation is not the most essential moment in these changes. The nuclear masses, when not of large size, may be regarded as identical with the naturally existing solitary glands.

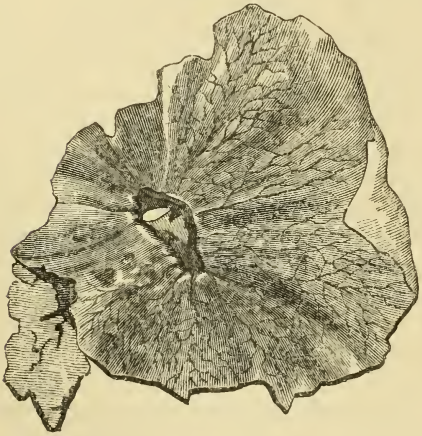
Croupy Inflammation resulting in so-called fibrinous exudation, which forms a false membrane, sometimes of regular areolar surface, is very rarely seen, at least in England, and is said by Rokitansky to be a "sequela or degeneration of exanthematic processes, as of variola, typhus, pyæmia, and particularly puerperal phlebitis. Sometimes inflammation of a low erysipelatous kind attacks the *submucous connective tissue* of the stomach, and occasions suppuration. The pus, after a time, escapes by numerous irregular cribriform openings into the cavity of the viscus."

Corrosive Poisons.—We proceed to notice the effects of caustic fluids, such as the mineral acids, which have been swallowed. The mucus in the mouth and fauces is coagulated into flocculent masses, the epithelium is detached here and there, and "converted into a thick greyish-white, rugose layer," and the subjacent mucous membrane is pale. If the caustic fluid has penetrated more deeply, "the superficial layers of the mucous membrane of the fauces and œsophagus are found congested, of a dirty, whitish, leaden hue, and the capillary network blackened by its carbonized contents. The lower strata of the mucous membrane, and the submucous cellular tissue, present serous infiltration. In the follicles at the root of the tongue, the mucous secretion is coagulated into dirty white masses. In a still higher degree of corrosive action, the entire mucous membrane is destroyed, and converted into a dirty grey mass, which is traversed by black vessels; the submucous cellular tissue is infiltrated, and partially ecchymosed; the muscular coat of the œsophagus itself is shrivelled, pale, ashy. In the highest degree, the mucous membrane of the œsophagus, together with the submucous cellular tissue, is converted into a soft, black mass, which is distended by a sanguinolent fluid, and is easily detached from the muscular coat. The latter is itself either destroyed in the same manner, or is perfectly colourless, friable, and presents an ashy, gelatinous appearance. The mucous membrane of the stomach," in consequence of being longer in contact with the corrosive substance, "almost invariably suffers the changes of the last degree but one, though in varying extent and thickness. It is either affected in single folds, or streaks which pass from the cardiac orifice to the lesser curvature, and from the large curvature to the pylorus; or over a large extent; or we find the entire surface converted into a black carbonaceous mass, of several lines in thickness, distended by sanguinolent fluid, and consequently presenting a tumefied appearance." The muscular coat is affected, and the parietes of the stomach are often perforated. "The acid affects the neighbouring organs through the membranes, and thus either coagulates or tans the contained fluids, fuses the tissues into a gelatinous mass, or carbonifies them; the discoloration produced is always very marked. In many cases, not only the blood of the neighbouring blood-vessels, but also of the larger trunks, and even of the aorta, is changed into pultaceous, pitchy, greasy, black cylinders. Beyond the stomach, and especially in the duodenum, and at the commencement of the jejunum, the effect of the lowest degree is exhibited in coagulation of the intestinal mucus, and of the chyle, in corrugation and opacity of the epithelium, in the tanned state of the mucous membrane, and the dark injection of its vessels." The highest degrees of corrosive action are rapidly fatal, "the lowest degrees are followed by exudative inflammation, the mortified epithelium sloughs, and being replaced by a new formation, as soon as the reaction has abated, recovery ensues." In all the higher degrees, inflammation, passing into suppuration, produces the separation of the superficial mortified layers. The suppurating process may be protracted, or may terminate early with the formation of cicatrices. "According to the depth to which the tissues are destroyed, the loss of substance is repaired under a formation of structures that vary in size and consistency." When the mortification is limited by the submucous cellular tissue the latter becomes condensed, and "forms, at some places, projecting ridges, or valvular, and even annular, duplications towards the œsophagus;" in this way peculiar membranous strictures are produced. "If the muscular coat itself is involved, it is partially or entirely destroyed, and the walls of the œsophagus are converted into a fibro-cellular firm tissue, which contracts, and thus produces the most important and most resisting strictures." Chronic suppuration sometimes occurs as the result of profound injury, leading to the formation of abscesses and sinuses of the muscular coat, and of the surrounding cellular sheath of the œsophagus. These may produce perforation of adjoining passages, the trachea or bronchi, or may heal, leaving considerable contractions of the tissues and strictures. Cicatrices and strictures are formed in the same way, though less frequently, in the membranes of the stomach. The morbid changes produced by arsenic are as follows:—"At one or more points to which the powder happens to attach itself to a larger amount, the mucous membrane appears plicated and tumefied, reddened, invested by a detached epithelium, and a tawny exudation; its tissue is softened, pultaceous; and at the spot where the white grains of arsenic are attached, it is converted into a yellowish or greenish brown slough." The tissue intervening between these solitary foci is often quite healthy.

ULCERATION OF THE STOMACH.

Perforating Ulcers.—Ulceration of the coats of the stomach is much less frequent than that of other portions of the intestinal canal. It occurs sometimes as the result of chronic gastritis, or of the corrosive action of poisons. These ulcers require no special notice, but there is one particular kind which is rather peculiar to this organ, and which is of especial interest, from its occurring in tissues which otherwise appear quite healthy, and from the serious and rapidly fatal effects to which it too often gives rise. Rokitansky terms this the perforating gastric ulcer, on account of its having a decidedly marked tendency to perforate the parietes of the stomach. He describes it as follows:—"In a well-defined case there is, in the region of the pylorus, a circular orifice of from three to six lines in diameter, with a sharp peritoneal edge, as if a round piece of gastric parietes had been punched out. When viewed from within, the loss of substance on the internal membranes of the stomach, and especially of the mucous layer, appears more considerable, so that the edges of the hole seem bevelled off from within outwards." In some cases the margins of the ulcer are quite smooth and thin, in others thickened and indurated. "The pyloric half of the stomach," Rokitansky proceeds, "is the seat of the ulcer: it is most frequently found in the middle zone of this portion; it is oftener seen at the posterior than at the anterior surface, almost always near to, and frequently at, the lesser curvature; and it occurs in extremely rare cases only at the fundus." A similar ulcer may form in the upper oblique portion of the duodenum, or in the lower end of the œsophagus, but not, as far as observation has yet shown, in any other part of the intestinal canal. The size of these ulcers, Rokitansky testifies, may equal that of a cheese-plate; we have never seen them exceeding that of

Fig. 131.



Perforating ulcer of stomach. The mucous membrane is puckered into folds round it.

a half-crown. Their form is commonly circular, at least in the outset, though they often become elliptical or quite irregular, as they extend. Sometimes the ulcer enlarges in its transverse diameters, so as to obtain a zonular form. Sometimes two ulcers coalesce together more or less completely. "In the majority of cases there is only a single ulcer," (there is no mention of more than a single one in three cases which are recorded consecutively in the Report of the Pathological Society for 1847-48,) "but frequently there are two or three, occasionally four or five, and these are then commonly placed above or near to one another at the posterior surface of the stomach, or at the lesser curvature." When the ulcer is perfectly circular, the narrowing of its area, as it extends in depth, is very marked, the muscular coat is less extensively destroyed than the mucous, and the peritoneum again less extensively than the muscular, the perforation, in fact, taking place, as Rokitansky describes it, in the centre of the included circle. The exact nature of the process by which the ulceration commences, has not yet been the subject of direct observation.

Causes of Perforating Ulcer.—The origin of these ulcers has been explained in various ways; the most important element in their causation must, however, be the corrosive action of the gastric fluid, since they are found only in parts which are exposed to its action, viz., the stomach and adjacent parts of the intestinal canal; but it remains to be shown how the tissues, which commonly possess the power of resisting digestion, are in this case affected by that process. It has been shown by physiologists that the chief reason why the stomach itself is not digested, is the circulation in its tissues of healthy blood, or, according to some, of alkaline blood. If the circulation is interrupted this immunity is lost and the gastric tissues become liable to digestion; and it is rendered probable by observation, that limited stasis, or stagnation of blood, always precedes the formation of such an ulcer; a dark red, brown, or black spot being (according to Förster) produced, which becomes necrotic, and is corroded by the gastric fluid. The stasis, or hæmorrhagic infiltration, may be brought about in several ways; either it may occur in the course of simple catarrhal inflammation, or be the consequence of extreme venous hyperæmia in cases of obstruction in the gastric or portal veins; or, again (as shown by Virchow), be caused by thrombosis or embolism of a small arterial branch. It is uncertain which of these causes is the commonest. The area of ulceration often corresponds to the distribution of one of the ultimate arterial twigs; nevertheless, embolism or thrombosis has only, in rare instances, been observed. We have twice observed arteries obstructed by a clot in the floor of gastric ulcers; but this fact seems to point rather to spontaneous arrest of hæmorrhage. Dr. Copland states, and some of our own observations are confirmatory, "that this affection is most frequent in needlewomen, or female servants. The patients, in most instances, have been anæmic, or suffering from disordered menstruation, as well as from pains in the stomach, but have generally been able to pursue their avocations, and to take their food, up even to the period of the fatal seizure."

Hæmorrhage and Perforation.—The ulcer, especially when seated near the smaller curvature, is apt to involve some arterial branch, from which blood is poured out in abundance. The hæmorrhage may be so copious as to destroy life at once, but more frequently death does not occur till after repeated attacks. The deeper the ulcer has extended the larger in general are the vessels it meets with, and the more serious, in consequence, the loss of blood. It has happened, that an ulcer, after perforating the coats of the stomach, has lighted upon the pancreatic duct and produced a fistulous opening into it. The most dangerous situation for an ulcer to occupy is the lower half or two-thirds of the anterior surface of the stomach, as, in case of perforation, there is no organ to which it can easily become adherent. On the posterior surface, adhesions form between the stomach and pancreas, or the adjoining lymphatic glands, and on the upper and pyloric part of the anterior surface the escape of the gastric contents into the peritoneal cavity is sometimes prevented by the left lobe of the liver. In a remarkable case, recorded in the Report of the Pathological Society, 1847-48, p. 252, the barrier opposed to the extension of an ulcer by the left lobe of the liver proved insufficient, as the destructive process continued until it perforated the diaphragm, and gave rise to hepatisation and a gangrenous cavity in the lower lobe of the left lung. These ulcers may heal at any period of their course; it is not uncommon to find the cicatrices, which are their results, on the inner surface of the stomach. There can be no doubt that a production of fibroid tissue is the means whereby the reparation is effected. This takes place both in the margins of the ulcer, producing thickening and subsequent contraction, and also at the base, which it lines with a thin smooth layer. Cicatrices of this kind present a depression of the size of the ulcer, surrounded by thickened and elevated margins; others, where more contraction has taken place, are of a linear or corded shape.

Hæmorrhagic erosion of the gastric mucous membrane is thus described by Rokitansky:—"There are round or roundish spots of the size of a pin's head or pea, or narrow elongated streaks, at which the mucous membrane appears dark red, lax, soft, bleeding, and presenting a depression in consequence of loss of substance or slight erosion. Sometimes this loss of substance involves the entire thickness of the mucous membrane and the submucous cellular tissue, and produces an appearance of small round or striated ulcers. This process is invariably accompanied by hæmorrhage," the effused blood being mixed in a more or less altered state with gastric mucus, which is poured out by the membrane affected with recent or inveterate catarrhal inflammation. The erosions are often very numerous, studding perhaps every part of the stomach, with the exception of the fundus; their chief seat is at the pyloric portion. They are not peculiar to any form of disease. Microscopic examination of one of these ulcers showed the surface sunk in, the basement membrane gone, and the tubes quite atrophied and replaced by low fibroid substance, infiltrated with diffused yellow pigment.

SOFTENING OF THE STOMACH.

This condition, once thought to be of great importance and to indicate disease existing during life, is now generally regarded as a change which takes place after death. Under some circumstances, as yet imperfectly understood, the gastric juice will act upon the walls of the stomach as upon the food during life, and partially or completely dissolve them. When the solution is only partial the mucous layer alone may be affected; its surface will be smooth without rugæ, the vessels containing blood will be stained black or brown, and the same colour may be seen diffused; while the whole wall will, of course, be thinner. The neighbouring parts, where the change is less advanced, will look pulpy and almost gelatinous. When the process extends more deeply, similar changes are seen to affect the muscular and fibrous coats, making them so soft as to give way with a touch, or actually dissolving them. It may happen that the walls, though not actually perforated, are so soft that the attempt to remove the organ causes its contents to escape into the abdominal cavity.

This change, whether partial or complete, is distinguished from conditions occurring during life, or from any possible result of the action of corrosive poisons by the following characters. The change is limited to that portion of the stomach in which food or acid liquid was contained; this, in the ordinary supine position of the body, will be the cardiac extremity and fundus, and especially its posterior part, so that, as Dr. Wilks points out, if the contents be removed, it may be possible to see, by the change which has occurred, to what height the food reached, the watermark being accurately defined by a distinct margin. Again, if perforation has occurred, a gradual thinning and softening of the coats will be observed all round, which will show that the opening cannot have been due to an accidental cut with the knife; while the entire absence of inflammatory reaction is sufficient evidence that the opening was not made, at least, long before death by ulceration or any similar process.

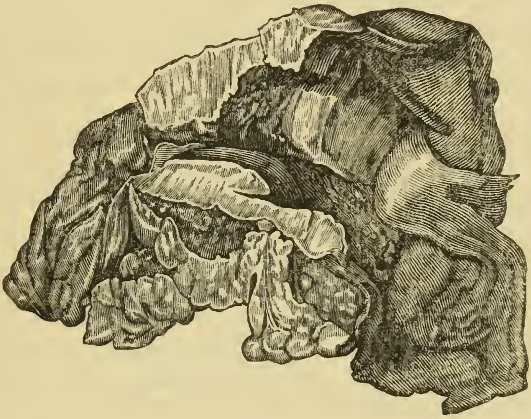
The softening may extend in some cases beyond the stomach to the lower end of the œsophagus, diaphragm, pleuræ, and lungs, or the œsophagus alone may be perforated, and the contents of the stomach escape into one pleura. Less frequently the change extends to the duodenum, where the mucous surface will be found pulpy and soft, or the whole wall destroyed.

Pathology of Softening of the Stomach.—It has been always held by most English pathologists, including Hunter and Baillie, that the appearances just described are solely due to cadaveric change. On the other, several continental authorities, as Cruveilhier and Rokitansky, have attributed them to a special disease, or have supposed that there were two forms of softening, that due simply to post-mortem digestion, and another due to disease, called *Gastromalacia*, and alleged to be specially prevalent in children. It has, however, been shown (especially by Elsässer) that the existence of such a disease is extremely improbable, and rests on no evidence; so that the original explanation of Hunter must be maintained. The alleged frequency of softening in children is probably to be accounted for by the excessive production of lactic acid in the acid fermentation of milk contained in their stomachs. The favouring conditions are, however, difficult to trace.

NEW GROWTHS IN THE STOMACH.

Fatty tumours, originating in the submucous tissue, and increasing in size, may either press inwards towards the cavity of the stomach, or outwards, towards the peritoneal sac. In either situation they may be sessile, or pedunculated. *Lymphoid* growths have been seen in cases of general lympho-sarcoma, or "adénie." *Fibroid nodules* sometimes form in the areolar submucous tissue, "chiefly in the vicinity of the cardiac orifice, and the lesser curvature." Tumours of smooth muscle-fibre have also been described. *Erectile tissue* may be developed at the free extremity of polypoid growths, or may occupy a larger surface of a sessile tumour. *Tubercle* is very rarely seen in the stomach, and only occurs in cases "where intestinal tuberculosis has advanced to an extreme degree." *Cancerous disease* of the stomach is frequently met with; this organ ranks next to the uterus in the list of mortality from this cause. Primary cancer exists in the majority of cases. Dr. Walshe speaks of secondary "as almost unknown," except where it invades the organ from extension of adjacent growths. One such case of secondary cancer has been recorded by Cohnheim. It is not uncommon to find a solitary growth in the stomach, no other part being implicated, probably in consequence of the fact that cancer attacking so vital an organ as the stomach causes death at a comparatively early period, before time has been given for extension to other organs. "The pylorus," says Rokitansky, "indifferently at all parts of its circumference, is known to be the chief seat of primary fibrous and areolar cancer of the stomach. From this point the degeneration extends chiefly along the lesser curvature over the pyloric half of the stomach; in many, though severe cases, it affects the entire stomach, attacking the fundus last, which, however, generally remains partially free. The parietes of the stomach may attain an inch in thickness, being rigid and generally tuberculated on their inner surface; the cavity of the stomach will at the same time be diminished in size.* In 120 cases collected by Köhler, fifty-nine occurred at the pylorus, seventeen on the lesser curvature, eleven on anterior and posterior surfaces, eight at the cardiac extremity, three on the greater curvature, three involving the whole stomach, and in one case at several points. Dr. Walshe states that cancer of the orifices may extend to the duodenum or the œsophagus. Rokitansky affirms "that cancer of the pylorus is accurately bounded by the pyloric ring, and hardly ever extends to the duodenum," whereas cancer at the cardia invariably involves a portion of the œsophagus. We certainly think that scirrhous disease of the pylorus does not extend much beyond its original site, at least along the intestine, although it may propagate itself to the head of the pancreas, or the adjacent lymphatic glands. Commonly, as Rokitansky describes it, the scirrhous pylorus is bound down by the degeneration of the tissues lying behind it, but in other cases it remains moveable, and may be felt as a distinct tumour having descended more or less over to the lower part of the abdomen. The pylorus, the cardiac orifice, the greater, and lastly, the lesser curvature are liable, according to

FIG. 132.



Scirrhus Pylori. At the diseased part the walls of the stomach are extremely thickened, and of a whitish colour.

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* Of this condition the following is an example:—A. M., æt. 63, dying with general chronic peritonitis and ascites. The stomach was very much contracted in all its extent, its coats were very much thickened, the muscular especially, the mucous membrane was apparently intact and thrown into folds. The muscular fibres were very evident and this tissue was greatly hypertrophied. Here and there, in the substance of it, nuclei and cell corpuscles were seen. These were, however, much more abundant in the submucous tissue, where they were very conspicuous. The new growth had also evidently infiltrated the mucous membrane and destroyed the tubes, of which only slight traces could be seen, the substance of the membrane being occupied by nuclei and corpuscles. The liver was small, contained no cancer, nor were there any enlarged glands.

the order in which we have placed them, to be the seat of cancer. Fibrous cancer, *i.e.*, scirrhus, undoubtedly is the most common, or, as we are inclined to think, a combination of scirrhus with colloid. Medullary cancer ranks next, according to Rokitansky,* and areolar or colloid last. He notices the frequent primary combination, and the yet more frequent secondary, of scirrhus with encephaloid, or of both with colloid.

Minute Characters of Scirrhus.—The following description was taken from an exceedingly well-marked specimen of scirrhus pylori, in which the walls of the passage were so thickened as to be nearly an inch in diameter. The cut surface presented a whity greyish tissue, contrasting well with the injected mucous membrane, and exhibiting a distinct striation vertical to the axis of the canal. A section under the microscope showed greyish white bands, separated here and there by transparent gelatinous matter. The bands consisted of homogeneous faintly-mottled substance, occasionally divisible into fibres closely resembling those of organic muscle, and, like them, exhibiting elongated nuclei when treated with acetic acid. Towards the mucous membrane this close stroma was replaced by a loose fibroid tissue, forming circular loculi of various sizes, which were filled with very various forms of cell-growth. Among them granule cells were often apparent, but the main mass consisted of nuclei and low developments of them. Some large mother-cells were seen, containing several well-formed nuclei and granulous matter: in the interspaces between the fibrous bands these mother-cells had attained a gigantic size, and appeared to constitute the loculi; one of them was distinctly bifurcated at its narrow end, and the branches were of some length. In this case, we consider that there was a combination of colloid with scirrhus, the former being constituted by the cellular substance. In Dr. Bennett's "Observation," xxi.,† the alteration which had taken place in "the walls of the stomach was wholly of a fibrous character." No cancer cells were detected, only elongated and fusiform nuclei; but they were numerous in the enlarged mesenteric and lumbar glands. This latter circumstance is, we think, decisive of the truly malignant nature of the morbid change in the stomach, and such a case may then be regarded as one of pure scirrhus.

Distinction from Simple Induration.—In such cases, where the scirrhus-like hardening of the pylorus does not present the minute characters of true cancer, the tumour may be either one of a different species, *e.g.*, a sarcoma, or else result from the process known as fibroid induration; nevertheless, it must be remembered that true scirrhus may, at a certain stage, exhibit over a considerable extent of its bulk, an entirely non-cellular structure, and it is only by examining other portions where the typical alveolar structure remains that we can get a true notion of its nature. The condition of the lymphatic glands is also important, since these are not affected in cases of chronic induration. Rokitansky enumerates, as distinguishing signs of cancer, the preponderating increase of substance in the submucous cellular tissue, and its want of uniformity, the accompanying cartilaginous hardness and closeness of texture, the fusion with the mucous and muscular coats, and particularly the alteration in the muscular tissue itself. We think the microscope, in practised hands, would generally clear up all doubt. When loculi of cell substance are mingled with the fibrous tissue, there can be little hesitation in regarding the growth as cancerous. If the structure is purely fibrous, attention must be directed to the limitation of the disease, and to the existence of the infiltrating, softening, and contaminating properties of cancer.

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* Förster, however, has found medullary cancer the most frequent.

† "Cancerous and Cancroid Growths," p. 46. Edinburgh, 1849.

There is, we are persuaded, a form of fibrous induration of the pylorus, accompanied by great hypertrophy of the walls and contraction of the orifice, which is quite unconnected with cancer. In this change the arterial branches are usually very atheromatous, and we have seen fatal hæmorrhage resulting from the rupture of such a vessel. Some authorities, as Dr. Wilson Fox, are disposed to regard these as cases of obsolete or degenerated scirrhus.

Medullary or Encephaloid.—The encephaloid form of cancer sometimes occurs as a sort of development of scirrhus, appearing as fungus or cock's-comb-like bleeding excrescences. Encephaloid, however, either in the form of knotted tumours, or degeneration of the submucous tissue, or infiltration of new-formed erectile tissue, sometimes occurs primarily. It forms soft whitish masses, with a tendency to softening and breaking down. The microscopical structure shows loculi, with numerous small cells, more uniform in size and shape than those of scirrhus, and, according to some observers, resembling those of the peptic glands. It is less confined to the pyloric extremity than scirrhus.

Epithelioma.—The ordinary flat-celled epithelioma, formed from pavement epithelium, does not occur in the stomach, except as an extension from the œsophagus, or more rarely from the mouth direct. This might be expected from the non-existence of pavement epithelium in the stomach. Cylinder-celled epithelioma (see page 180), on the other hand, sometimes occurs and appears to be developed from the normal epithelium of the stomach. It resembles encephaloid in its naked-eye appearance, but gives not so much a creamy juice, as plugs of softened matter (comedones), like those of the other form of epithelioma. It is usually found in a belt round the pylorus, like scirrhus, but shows fungous or polypoid outgrowths on the mucous surface. Extension to other organs and reproduction in distant parts have been observed.

Colloid cancer, affecting the stomach, behaves much as it does elsewhere, it originates, as the other species generally do, in the submucous tissue, and, as in a case excellently described by Dr. Walshe, may cause atrophy and destruction of the mucous membrane, over a more or less considerable space.

Changes in surrounding parts.—The mucous membrane covering the cancerous growth may undergo various changes. "It sometimes degenerates into an areolar cancerous tissue, which discharges large quantities of gelatinous mucous fluid; or it is converted into erectile tissue as a fungoid growth, which becomes the seat of encephaloid infiltration, suppurates, and partially exposes the submucous scirrhous cellular tissue; or, lastly, it most frequently becomes the seat of a sloe-black softening, with hæmorrhage," or it is quite destroyed, and the sloughing process attacks even the denuded scirrhus itself. In a specimen we recently examined of scirrhus pylori, where the mass, limited to the pyloric region, was exposed on its inner surface, forming a sloughy ulceration with elevated, thickened margins, a fatty transformation had very evidently commenced. It was most apparent in the contents of the loculi, which in some parts consisted of well-formed nuclei and granulous matter, but in many others only of an amorpho-granulous substance, imbedding much oily matter. It is conceivable that the further progress of this change might have effected a cure. In this case it was very distinct, and is worthy of remark, that while the muscular coat had undergone very considerable hypertrophy, it was in no degree affected by the cancerous disease. This, though encircling the pyloric outlet, had not caused any actual obstruction to the passage, nor was the stomach distended in any very considerable degree. The cause, therefore, of the hypertrophy of the muscular coat does not seem sufficiently explained; but it is very commonly seen in cases of scirrhus.

A case is recorded by Andral, in which enormous dilatation of the stomach had taken place, although the pyloric orifice was free and even larger than natural. He accounts for this by the non-existence of muscular fibres in this instance in the vicinity of the pylorus. Admitting this explanation, we are inclined to think that the very alteration of the natural condition of the outlet, its being reduced to a passive and rigid orifice, may necessitate a greater exertion of the muscular fibres, while, if it fails to take place, and thus to induce a conservative hypertrophy, dilatation must result. Dr. Walshe, after mentioning the more usual occurrence of dilatation ensuing when the pyloric opening is obstructed, and contraction when the cardiac is, the size remaining unchanged when the body of the organ alone is affected, notices as "less intelligible" the fact to which we have just referred. He also remarks that, "as a general truth the mucous membrane exhibits a notable power of resistance to the encroachment of the disease." This, we think, is true, at least as far as naked-eye investigation can ascertain; but in one case, where the mucous membrane appeared tolerably healthy, we found the tubular secreting structure in process of disorganization, not, however, from the extension of the cancerous disease.

Ulceration, usually the result of secondary gastric cancer, may cause perforation of the stomach and fatal peritonitis; it more frequently happens, however, that the effusion is prevented by the formation of adhesions between the threatened part and contiguous viscera. The liver and pancreas may thus become the seat of further cancerous invasion and destruction, or the ulcer may eat its way into the transverse colon, and thus cause an unnatural communication between its cavity and that of the stomach. A dark fluid, resembling coffee-grounds, is often found in the cancerous stomach after death, as well as vomited during life. In one case, where we examined it, we found it to consist of very numerous blood-globules, together with black granules and grains (probably altered hæmatine), and a very large quantity of amorphous, with some oily matter. It is to be remembered that vomited matter of this kind is not peculiar to cancerous disease, the same may be brought up when there is simple exhalation of blood from the mucous membrane, common ulceration, or follicular ulceration, or even softening. The only circumstances necessary for its production are hæmorrhage and the acid secretion of the stomach.

Sarcomatous or fibroid tumours sometimes occur in the stomach, but not commonly. A spindle-celled sarcoma, with much intercellular substance, and giving no cancerous "juice" on section, is recorded by Dr. Cayley (Path. Trans. vol. xx. p. 170); a similar case was observed by Dr. Wilks (Ibid. vol. x. p. 146).

Other morbid conditions of the mucous surface are *pigmentation*, which is extremely common, and usually, as we have said, connected with chronic inflammation, perhaps with ecchymosis; *calcareous deposit* in the mucous membrane, observed in one or two rare cases by Virchow; and *lardaceous* (or amyloid) *degeneration*, very rarely observed in some cases of this disease.

Abnormal Contents of the Stomach.—Besides blood there may be several other matters abnormally present in the stomach. Unhealthy mucus in large quantities, purulent and other exudations, bile, biliary calculi, fæcal matter, and lumbrici, are more or less often met with. Foreign bodies of the most various kinds are also to be included in the list, as the sealing-wax, brick-dust, cinders, &c. swallowed by hysterical females, or those who are subjects of the morbid state termed *pica*, or by actual lunatics. A remarkable case of this kind has been recorded by Mr. Pollock, in the Report of the Pathological Society for 1851-52, in which the stomach was distended by a large mass of hair and string, while another occupied the lower portion of the duodenum and commencement of the jejunum.

Various vegetable parasites sometimes occur. The yeast plant (*torula cerevisiæ*) is generally, if not always, found in cases of diabetes. Leptothrix filaments are perhaps derived from the mouth. Aphthæ sometimes form considerable layers, as on the mouth of children in thrush. They appear to be generally found in children, but also in adults who have taken much milk; and in other cases without any reference to food. The sarcina ventriculi (see page 207) is probably not the cause of any special disease, but found in most cases of dilatation and obstruction; sometimes in enormous quantities.

VI.—ABNORMAL CONDITIONS OF THE INTESTINAL CANAL.

Congenital Malformations.—The intestine is not unfrequently *defective* in some part of its course; this most commonly is the case near its lower termination, and involves an imperforate condition of the anus (atresia ani). Sometimes the intestine is only unusually short and of uniform calibre, or consists of several detached cæcal portions, or it may terminate at the umbilicus, or in a cloaca common to it and the genito-urinary organs. Andral refers to a case in which there was only a single straight canal, extending from the termination of the œsophagus to the commencement of the rectum, to another in which the duodenum was double, a third in which there were two colons, to a fourth in which the appendix vermiformis was unusually large, and at the same time double. All these, except the first, are instances of *excessive* development, though Rokitansky refuses to regard them as such, and considers them as "arrests of formation."

Atresia ani is the most important of these malformations. Sometimes we find absolutely no anal orifice, sometimes one which merely leads into a short cæcal pouch; the colon in both cases ending blindly in a mass of connective tissue. The rationale of this defect evidently is, that the inferior extremity of the rectum, formed by inversion of the external integument, fails to become connected with the colon, which is formed out of the third embryonic layer.

The mode of development of the intestine gives occasion for some of its congenital abnormalities. The rudimentary intestine consists of a portion pinched off from the vitelline vesicle and the communication between them, the omphalo-mesenteric or vitelline duct remains open for a considerable time. If the opening remain after birth, there may be a fissure of the abdominal walls at the umbilicus into which the ileum will open either directly, or by a channel given off laterally from it, which is the remnant of the omphalo-mesenteric duct. Or, again, the walls may be completely closed, but the structure last mentioned may remain in the form of a *diverticulum* either attached to the umbilicus by a cord representing the rest of the duct, or hanging free into the peritoneal cavity. The latter case, in which the original duct is represented by a diverticulum, or blind appendage, of the intestine, is the most common, and is of some importance.

Diverticulum Ilei.—This structure is found on the lower part of the ileum, from one to three feet above the ileo-cæcal valve. It is from half an inch to six inches long, resembling the intestine in structure, with its walls, composed of all the three coats, and cylindrical or very bluntly conical in form; its cavity, equal to, greater or less than that of the intestine. It usually proceeds from the convex side of the intestine, or that remote from the mesentery. The explanation we have given of this curious structure was first suggested by Meckel, whose views, after much controversy, are now generally accepted. When the extremity of the diverticulum still remains attached to the umbilicus by a fibrous cord, this may give rise to strangulation of a loop of intestine and fatal obstruction. Four specimens, showing obstruction from this cause are preserved in the Guy's Hospital Museum; and one is described by Dr. Wilks in the "Pathological Transactions" (vol. xvi. p. 126). Fatal ulceration and perforation of such an appendix have been seen in a case of typhoid fever. A case is recorded by Dr. Lionel Beale, in the Report of the Pathological Society, for 1851-52 (vol. iii. p. 366), in which fatal peritonitis ensued from softening and perforation, caused by the lodgment of a cherry-stone and other foreign bodies, of the lower part of a diverticulum, which was twice as broad there as in the upper part adjoining the intestine. It is very conceivable, therefore, that these offsets, like the vermiform appendix, may become sources of danger by offering a favourable situation for the lodgment of cherry-stones, or other indigestible matters.

False Diverticula.—Since somewhat similar cæcal appendages are found in other parts of the intestine, a distinction has been drawn between true and false diverticula. The latter name is applied to partial dilatations, or herniæ of the mucous membrane covered by the serous coat, such as occur also in the bladder. Rokitansky gives the following description of the characters of "false diverticula," which he regards as mere herniæ of the mucous membrane, "resulting from the separation of the fibres of the muscular coat." They consist solely of mucous membrane and peritoneum. They occur at any part of the small and large intestines. "They are found in considerable numbers. They occur from the size of a pea to that of a walnut, in the shape of round baggy pouches of the mucous membrane. They form, more especially in the colon, nipple-shaped appendages, which occasionally are grouped together in bunches; when occurring in the small intestine, they are commonly developed on its concave side, and are therefore placed between the layers of the peritoneum; when in the colon, the fæces are retained by them, and dry up into stony concretions." This condition may give rise to ulceration and fatal perforation; as in a case recorded by Mr. Sydney Jones, where a communication was thus formed between the sigmoid flexure and the bladder. ("Trans. Path. Soc." vol. x. p. 131). In this case a fæcal concretion from the false diverticulum was actually found in the bladder.

Dilatation.—Uniform dilatation of the intestine may take place either from inaction of the muscular coat, or from distension from accumulation of its contents above a stricture. Disease of the nervous centres, inflammation of the serous investments, or simple atony of its contractile fibres, may be the cause of paralytic inaction and consequent distension. When a stricture exists, enormous dilatation sometimes takes place. Andral relates a case in which the large intestine was so distended that it resembled that of a horse, and concealed almost all the viscera. In such cases the muscular coat may be in full and painful activity, not ceasing its action, although unable to overcome the obstruction, but reversing the current (axial) so as to produce fæcal vomiting.

It is singular that some of the most extreme cases of dilatation are those in which there is no obstruction; as the remarkable case described by Dr. Peacock, ending in fatal constipation where the ileum became gradually distended to six or eight inches in diameter ("Trans. Path. Soc." vol. xxiii. p. 104). A greatly dilated intestine is never quite normal in structure; sometimes the muscular coat becomes hypertrophied, sometimes all the coats are thinned; and very often the mucous coat is ulcerated. We have nearly always found the latter change both in simple passive dilatation and in that arising from obstruction; and this, rather than violent rupture, is most often the cause of the bowel giving away.

Contraction of the intestine may occur either throughout a considerable extent, or in a very small one. The first is not a condition of disease, though it has been mistaken for such, and results merely from the canal at that part being empty for some time; so that the natural contracting efforts of the circular fibres are unopposed, and the sides of tube are brought together. We have seen the descending colon in this way so shrunk as to be scarcely larger than the little finger. Of course this condition is most likely to occur in the part of the intestine below a stricture. The second kind of contraction of the intestine is almost always morbid, and may result either from external pressure, as from a strangulating band, or compressing tumour, or from disease of the tissue in the part affected. The cicatrix of a simple or tuberculous ulcer, which has assumed an annular form, or a cancerous growth of like shape, has in most instances been the cause of stricture originating in the intestine itself.

Changes in the position of the intestine leading to morbid effects, require some particular notice. The most common of these constitute the several varieties of hernia. As these are fully described in all surgical works, it does not seem necessary to make mention of them here; and we shall therefore confine our attention, following Rokitansky, to the morbid changes of position which befall the intestine within the abdominal cavity. A portion of intestine may be twisted upon its own axis, so as to obstruct the canal by the approximation and contact of the walls. This seems to have been observed only in the ascending colon. The mesentery may be twisted upon itself, forming a kind of cone, with more or less of the intestine attached to its base, which becomes strangulated by being twisted round the mesentery. "One portion of the intestine, either single or double—a coil—may afford the axis round which another portion with its mesentery is thrown, so as to be throughout in contact with the circumference of the axis, and thus to compress it like a ferule. A coil of small intestine, the sigmoid flexure, or the cæcum, may form the axis." Abnormal length of the mesentery probably predisposes to these affections. More frequent than the foregoing, are the instances in which a portion of intestine, generally the small, becomes strangulated in consequence of having got into one of the following situations: (1) into the fissure of Winslow; (2) into an opening in the mesentery; (3) into an opening in the omentum; (4) into spaces included by corded bands of false membrane, and various parts of the abdominal viscera; (5) into similar spaces formed by a long vermiform appendage, or intestinal diverticulum.

The colon and the rectum have been known to be compressed and obstructed by a mass of loaded small intestine lying upon them; and Andral refers to a curious case in which the transverse colon, in a child of six months old, was compressed between the duodenum and the vertebral column. These varieties of incarceration occur at every period of life; they are more common, Rokitansky avers, in the female sex than in the male, because the sexual organs of the former not only offer an additional point of attachment for constricting bands, but may also themselves give rise to constricting growths. Thus it is not very uncommon to find a coil or coils of ileum attached in the pelvis by fibrous adhesions resulting from metritis or parametritis; and we have seen several cases of fatal strangulation depending on this cause alone. The consequences of strangulation taking place in any of the above ways are distension of the intestine above the compressed part, peritonitis and ileus. The incarcerated portion in the cases where there is much pressure on the mesenteric vessels, is peculiarly liable to congestion and gangrene. Andral well remarks that the mere existence of the bands of adhesion, forming the orifices above described, by no means necessarily involves strangulation of the intestine; this, however, in such cases, may come on very rapidly when the included portion of intestine becomes distended from any cause. In some cases symptoms are observed for several months or years, indicating that some impediment to the free passage of the intestinal contents exists.

Dr. Peacock has related in the Report of the Pathological Society, for 1848-49, two cases of so-called mesocolic hernia; the small intestines being contained in a sac, formed by the layers of the meso-colon. In the first of these cases there was no strangulation, though the ileum passed out of the sac over "a thin crescentic edge;" in the second the ileum was strangulated at the part where it escaped.

Wounds and lacerations of the intestines demand a brief notice.

When the bowel is wounded by the thrust of a cutting instrument, the danger of escape of the intestinal contents, and of consequent peritonitis, is not so extreme as might be supposed. The mucous membrane being somewhat loosely attached and thrown into natural folds, protrudes at the orifice so as to close it, if it be not very large; at the same time the constant pressure of the parietes tends to keep the wounded point applied against the opposing surface, to which, moreover, it becomes glued before long by adhesive exudation. If an opening is made into the intestine by ulcerative perforation of its wall, the effusion of lymph uniting it to adjacent parts becomes occasionally the medium through which a fistulous communication is established, either with the exterior of the body, or with another hollow viscus in the vicinity. Sometimes the ulceration instead of penetrating further, causes simply the formation of abscess in the subperitoneal cellular tissue, fæcal abscess, as it is termed. When the intestine is divided completely across, the two ends will occasionally unite, if the serous surfaces be brought together and maintained in apposition by stitches, but no one has yet succeeded in repeating Ramdohr's experiment (said to have been successful), in which a large portion of an intestine having mortified, was cut off, and the upper end inserted within the lower and kept in that position by a suture. It is very unlikely, that a mucous and a serous membrane would unite by adhesions, which is said to have occurred in that case; the serous covering alone, and not the mucous surface being disposed to adhesive inflammation. The intestine may be ruptured or even completely torn across, by a blow on the abdomen without any laceration of the walls of the body; and sometimes even without any external bruise. The sudden escape of the intestinal contents produces acute and violent peritonitis.

Invagination of the intestine implies the inversion of a portion, and its intrusion into another, an upper portion being generally intruded into a lower, and the converse rarely (Dr. Brinton thought never) occurring. The following account we abbreviate from the very full details given by Rokitansky. Invaginations not uncommonly form during the last moments of life, especially in diseases which give rise to irregular and disturbed innervation. Thus they are often met with in the dead bodies of children who have died from hydrocephalus. They are characterized by the absence of all appearances of re-action, such as we shall presently mention, and by the parts being easily restored to their proper situation. Several often occur in the same case. When they occur during life as a primary affection, or consecutive only to diarrhœa, they speedily bring the patient into a condition of extreme danger. Every intus-susception must present at least three layers, as will be readily comprehended on viewing the accompanying diagram; the outer, called the sheath, is formed by a portion of intestine in its natural position; the middle is formed by the portion of intestine immediately above, which is inverted; so that its mucous surface looks outward, and is in contact with the mucous surface of the sheath: the inner is the portion of intestine next above, with its serous surface opposed to that of the middle layer, which is simply intruded into the canal beyond it. Five layers will exist if another portion of intestine be forced down, and inverted into the original intus-susception, which then becomes a sheath to it. "Between the middle and inner layers, there is a portion of mesentery corresponding in size to that of the intestine displaced, and folded up so as to represent a cone, the apex of which lies at the free termination of the volvulus, with its base in the sheath, and at the entrance to the invagination. This portion of mesentery is always in a state of tension, which chiefly affects the part belonging to the inverted tube, and has a singular influence upon the form of the volvulus. It is the cause of the following circumstances:—First, that the volvulus (the middle and inner layers) does not lie parallel to its sheath, but always offers a greater curvature than the latter; the inverted tube (the middle layer) being compressed in its concavity into tense transverse folds. Secondly, that the orifice of the volvulus does not lie in the axis or in the centre of the sheath, but external to it; and that following the traction exerted upon it by the mesenteric fold that belongs to the inverted intestine, it is directed towards the mesenteric wall of the sheath; that it is not circular, but represents a fissure. Intus-susceptions occur with equal frequency in the colon, and small intestine; but several cases occurring in the former are remarkable on account of the magnitude they attained. In these the sheath contained a very long portion of the colon and ileum; both may be inverted two or three times, and the intus-suscepted part advances to the vicinity of the anus." The ileo-cæcal valve has even been seen protruded at the anus.

Fig. 133.

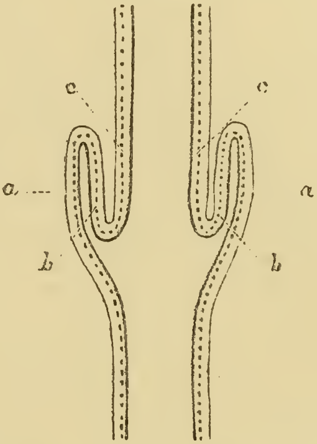


Diagram of intus-susception.

a, The sheath.
b, The receding or inverted tube.
c, The entering tube.

Causes and Consequences of Invagination.—Contraction of one part and paralytic distension of another, are probably the conditions which give rise to invagination; the upper contracted portion, whose muscular coat is for some distance upwards in a state of activity, being propelled onward into the dilated part below. Disordered and irregular innervation is probably the remote cause, in this as well as in the case of intus-susceptions taking place during the agony. Diarrhœa is the most frequent predisposing cause; but, in some cases, a tumour growing into the intestinal cavity occasions invagination. Increase of the invagination always takes place by more and more of the intestine becoming inverted, so that the upper border of the middle layer is continually shifted lower down. The consequence of invagination is, as may be expected, peritonitis and its results. Inflammatory congestion is set up, not at first, in consequence of annular constriction of the volvulus, but from compression of the vessels; especially the veins of the portion of mesentery which is dragged down by the advancing and inverted layers. This obstruction to the circulation, Rokitansky says, gives rise to violent inflammation, with "plastic effusion on the contiguous serous surfaces of the entering and receding (inverted) tube. The inverted portion is invariably the one that suffers most;" "and it is characteristic, that even when the inflammation of the volvulus (or entering portion) runs high, its mucous membrane remains pale." Even the sheath is not so much affected in smaller invaginations. The tumefaction resulting from the inflammatory congestion may cause strangulation of the volvulus, usually at the entrance, but sometimes at other points. If death does not result from the peritonitis, the ensuing gangrene, or the strangulation, recovery takes place with one of the three following terminations to the morbid process:—(a) Complete adhesion having been formed between the two opposed serous surfaces of the sheath, and the inverted tube at the point of inversion, the whole contained volvulus mortifies and is thrown off, and becomes discharged *per anum*. In a case referred to by Andral, the portion of intestinal tube thus evacuated measured eighteen inches; and Hévin relates two cases, in one of which twenty-three inches of the colon were thus parted with, and, in the other, twenty-eight inches of small intestine. An annular swelling, more or less interfering with the canal of the intestine, is found in the corpses of persons in whom this termination has occurred, besides adhesions of the serous surfaces in the vicinity. (b) "In rare cases, in which the incarceration has been developed at an unusual point, only a partial sloughing of the volvulus takes place; and the portion which lies above the strangulation is retained. Under these circumstances, the latter forms a conical plug with a narrow channel, and projects into the cavity of its sheath, surrounded by a thick fringe of mucous membrane." (c) Occasionally, after adhesion has taken place, the inflammation abates, and the volvulus is retained. The first mode of termination is the only one that produces a permanent recovery; in the others there always remains a degree of chronic hyperæmia, with liability to exacerbation. No age is exempt from the occurrence of invaginations. Andral quotes from Monro a case of a very considerable one, in a child four months old.

Prolapsus ani is an affection very analogous to invagination, differing chiefly in not having a sheath, as Rokitansky remarks, and also, as we think, in the peritoneum being less involved. In trifling cases, only a fold of the mucous membrane comes down, but, in the more serious, both the mucous and muscular tunics descend. The tumour thus formed is of a sausage or pyriform shape, pediculated by the contraction it undergoes at the anus, and having at its extremity in the minor degrees a round central orifice, and, in the greater, an eccentric fissure. The results of prolapsus ani (more properly *recti*), are not, for the most part, nearly so serious as those of invagination. In some cases, indeed, strangulation takes place, the everted part swells to twice or thrice its proper size, assumes a red, purplish colour, with an appearance of ecchymosis and of impending gangrene. More often, even in inveterate cases, there is only a discharge from the irritated or mucous membrane of a thin muco-sanguineous fluid, with, perhaps, some superficial ulceration. In some cases, the epithelium of the mucous surface, from constant exposure, assumes a cuticular character. The affection is of common occurrence in children, for which Sir B. Brodie assigns the following reason :—"The attachment of the rectum to the surrounding parts does not extend so high in children as in persons of mature age; while the reflection of the peritoneum takes place lower down; and hence the rectum is more liable to be pushed out." There must also be relaxation of the sphincter ani, which, if contracted, will prevent prolapsus.

INFLAMMATION OF THE INTESTINE.

The muscular coat of the intestines is very rarely, if ever, primarily the seat of inflammation, though it very often is involved by extension of the mischief from the serous or mucous tunics. We have, therefore, now to consider chiefly the condition of the mucous membrane and its follicles when inflamed. It must be premised that no kind or amount of vascular injection can be accepted by itself as a decisive proof of the existence of inflammation. Obstruction to the free return of blood by the veins, according to the degree in which it exists, will produce ramiform, patchy, or general injection; and the same cause will also give rise to the punctiform, which has been thought more characteristic of active hyperæmia. Very marked injection also results from mere gravitation of the blood after death to the most depending parts. MM. Trousseau and Rigot found in dogs, which had been suspended after strangling in a vertical position, the blood collected in those parts of the intestines which were the lowest, giving the mucous membrane and its villi a deep red tinge, and extravasating itself on the surface and in the submucous tissue. When the bodies were suspended in the reverse position, the same effects took place in those that were then the lowest. This hypostatic congestion is especially liable to take place in fevers, in severe cases of which the blood is so commonly found fluid after death. Redness, resulting from mere staining of the tissues, is sometimes very similar to that of active hyperæmia. The injection which takes place from engorgement of the veins may be distinguished from that of inflammation, by the circumstance of its being always traceable to distended veins; and, further, we think, by the redness being more general than that of inflammation commonly is. As a rule, the smaller and more isolated the patch of injection is, the more likely is it to be inflammatory; thus we find small separate patches of injection around commencing typhoid deposits, which contrast with the generally pale mucous membrane.

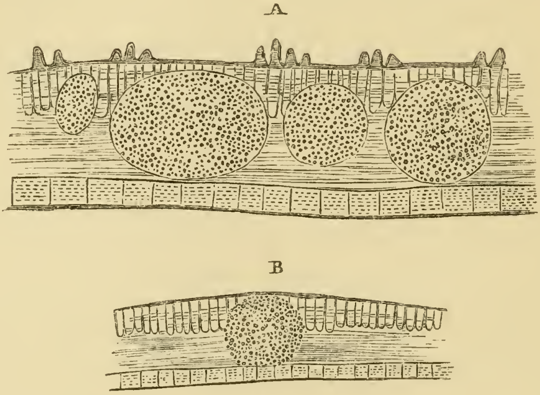
Catarrhal Inflammation.—Inflammation, affecting chiefly the mucous lining of the intestines, was distinguished by Cullen as *enteritis erythematica.* It corresponds to the muco-enteritis of others, especially the French pathologists. Rokitansky speaks of it as catarrhal inflammation, which may be either acute or chronic, and either attack the mucous membrane uniformly, or be developed mainly in the villi and the follicles. It is excited by various causes of irritation, and especially by certain atmospheric influences. "The anatomical signs of the *acute form* are, more or less intense redness and injection of the mucous membrane, affecting its entire surface, or appearing as punctiform reddening from affection of the villi, or as a vascular halo surrounding the follicles; relaxation of the tissue and intumescence of the mucous membrane equally affecting the entire substance, or only the villi and follicles; opacity of the mucous membrane and its epithelium, from infiltration of the former and softening of the latter; friability and softening of the mucous membrane. The submucous cellular tissue is injected, relaxed, and infiltrated with a watery, opaque fluid; the secretion is at first copious and serous; as the affection increases in intensity the former diminishes in amount, becomes opaque, viscid, and puriform.

Chronic Inflammation is characterized, in addition to the above signs, by a dark, rusty, livid discoloration, which in severe cases appears to pervade the entire mucous membrane; by a tumid state of the mucous membrane and its follicles, accompanied by increased density of the tissue, with copious secretion of an opaque greyish-white, or yellow puriform mucus. The acute form may subside completely, or several relapses occurring it may merge into the chronic, which seldom "admits of a complete cure." The following are the principal changes produced by chronic intestinal catarrh. (1) A more or less abundant deposit of black pigment in the whole mucous membrane, or in its villi or follicles only. (2) A permanent tumefaction of the mucous membrane, depending probably on dilatation of its vessels and interstitial exudation, which causes increased density of its tissue, and may give rise "in higher degrees to elongation of the membrane, and formation of folds and polypi." (3) Hypertrophy of the submucous cellular tissue and of the muscular coat. (4) Profuse secretion of a greyish-white and milky, or of a transparent gelatinous and viscid mucus." Suppuration and ulceration sometimes result from catarrhal inflammation, especially when an acute attack supervenes upon the chronic form. "The mucous membrane is converted into a dark-red, granulated, and friable tissue, on the surface of and within which suppuration is established." The burrowing of the ulcers, so as to pass through even the muscular coat, gives rise to sinuses, in the vicinity of which there is often a production of polypoid growths from the mucous membrane. Corrugation of, and pigmentary deposit in, the tissues, are constant accompaniments of this process. Cicatrization of the ulcers and sinuses takes place, with formation of the usual fibroid tissue, which by its subsequent contraction may give rise to puckering or obstruction.

Parts affected by Catarrhal Inflammation.—Rokitansky states that the most usual seat of catarrhal inflammation is in the cæcum and rectum; we doubt if this is true, if applied both to the acute and chronic forms, for there can be no doubt that, in the majority of instances of muco-enteritis, the small intestines are affected even to a greater degree than the large. He observes himself "that catarrhal irritation, and even inflammation, undoubtedly often affect the duodenal mucous membrane, and are frequently induced by an anomalous condition of the bile." This condition, as Dr. Stokes has observed, "may extend to the biliary ducts and give rise to jaundice." Besides the tumefaction of the mucous membrane, the increased pouring out of mucus, and the enlargement of the glands of Brunner in this situation, we have noticed an atrophy or destruction of the villi, which had lost their sharp exterior margin of limitary membrane, and were shreddy and wasted. The chronic form of catarrhal inflammation does seem to be more frequent in the large intestine; at least, the slaty and black discoloration is more often seen in the cæcum than elsewhere, but it may admit of some doubt whether this discoloration is a certain sign of previous inflammation, whether a deposit of pigment may not take place here as in the areolar tissue of the lung, or in the skin, without any disease. We have certainly examined instances in which the microscope showed nothing the least abnormal, except the pigmentary deposit.

Affections of the Intestinal Glands.—It seems very doubtful whether there is a distinct affection such as that which has been termed glandular or follicular enteritis. Dr. Copland speaks of it as occurring almost always consecutively to other diseases, as fevers continued or remittent, dysentery, and even tuberculosis. Rokitansky does not seem to recognize its special character, but to consider that the follicles may be more particularly affected in morbid processes of different kinds. In this opinion we entirely coincide, but wish to notice here certain points in the anatomical structure of these parts, which throw light on their pathological changes. The solitary glands of the intestine, which occur both in small and large, as well as their aggregations constituting the patches of Peyer, which do not extend beyond the ileo-cæcal valve, are quite destitute of the follicular character, that is, agree only in name with those glands which are involutions of the general mucous surface, invested by a lining of epithelium. They are simply solid aggregated masses of nuclear particles and lymphatic corpuscles, imbedded in a nucleated stroma, with very little intervening granulous matter, and lying completely beneath the sheet of basement membrane that covers the surface. They are not contained in a distinct capsule of homogeneous membrane, but lie partly in the corium of the mucous tissue, partly in that layer of nuclei and granulous matter which is spread under the basement membrane, and forms the chief substance of the villi, to which we gave some years ago the name of "substratum." It is apparent from this structural arrangement that they cannot be secreting organs like the Lieberkuehn tubes all round them, for they have no outlet. On the other hand, they are from their very structure peculiarly liable to become enlarged and prominent, the nuclei attracting to themselves plasma, and developing into cells, so that the actual number of lymph corpuscles is very greatly increased. There seems no doubt that they are much more developed in some persons than in others, and we are inclined to think that masses of precisely similar appearance may be formed in the mucous membrane solely as the result of irritation. We have been led to this belief from having found only a few distant and

FIG. 134.



Vertical section of Peyerian patch, and solitary gland of large intestine. The glands in both are rather enlarged.

A. Peyerian patch from ileum.
B. Solitary gland.

widely scattered glands in the mucous membrane of some persons, even when it was examined after immersion in acetic acid, which makes them much more opaque and white, while in other cases they were extremely numerous and close together.

These structures, both the solitary and agminated glands, are liable to hypertrophic and hyperplastic changes in various morbid conditions of the mucous membrane, which changes are liable, in certain diseases, to end in degeneration and necrosis. Simple inflammation, chronic or acute, of the mucous surface, leads to enlargement chiefly of the solitary glands. This may go on to sloughing and ulceration. In tuberculosis of the intestines the latter changes are the most conspicuous, but here also there is at first a stage of proliferation and enlargement. In some of the specific fevers, especially in typhoid, a similar series of changes is seen, chiefly affecting the Peyer's patches. It does not appear that the process ever precisely resembles suppuration. Dysentery is accompanied first by enlargement, then by sloughing of the solitary glands of the colon.

Follicular Ulceration of the Colon.—We shall now subjoin Rokitansky's account of "ulcerative inflammation of the follicles of the colon, such as we find in lientery, brought on by tedious diarrhœas. The follicles are at first tumefied in various degrees, and consequently project as smaller or larger, round, conical nodules on the internal surface of the intestine, being surrounded by a dark red vascular halo. Ulceration now ensues in the interior of the follicle, the small abscess penetrates the mucous membrane within the vascular halo, and a fringed ulcerated opening of the size of a millet-seed appears, which leads to a small follicular abscess with red spongy walls. The ulceration continues, and the follicle is eaten away," so that an ulcer of the size of a pea or lentil is formed. In its further progress the mucous membrane becomes extensively destroyed, and that with great rapidity, the muscular coat being frequently exposed. The most extensive destruction always takes place in the sigmoid flexure, and in the rectum. The disease is always confined to the colon, but when it runs a very rapid course it may be accompanied by catarrhal irritation of the small intestine. We have notes of a case in which there were a great number of the so-called solitary follicles in the lower part of the small and in the large intestine, although no irritation had existed during life. Their development seems to depend on some other cause besides mere irritation, though this, no doubt, promotes it. It can scarcely be supposed that the Lieberkuehn follicles, which are mere involuted extensions of the mucous surface, should not be affected wherever the mucous membrane is, but the solitary glands, which differ essentially in structure, seem to be by no means necessarily involved.

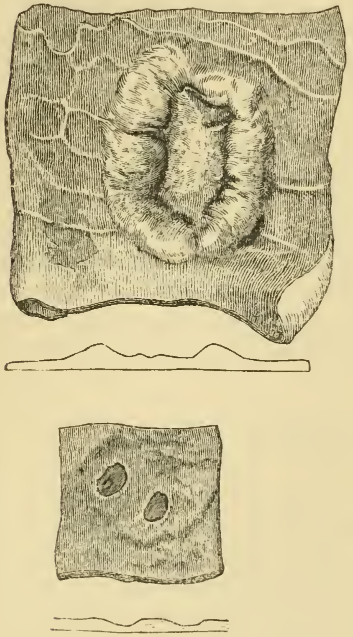
Phlegmonous Inflammation.—We proceed to the rare form of enteritis, termed by Cullen E. Phlegmonodea. This, if it deserves the name of a distinct variety of inflammation, should be described as inflammation of the intestinal walls, since the changes of the mucous membrane are not characteristic. The effects produced by this, which, indeed, are just those of common inflammation, have already been several times adverted to; but we think it well to subjoin the following excellent description from the pen of Dr. Copland:—"The villous coat in acute enteritis is not only more vascular and turgid, but it is also softer, and sometimes thicker than natural. If the inflammation has proceeded far, it presents a brick-red tinge, and is easily detached from the subjacent coats, the connecting cellular tissue being soft, turgid, and inflamed. When this state exists in a considerable portion of the tube, the coats are apparently thickened, arising from the extension of the inflammation to the more external tissues, till the attached surface of the intestinal peritoneum is reached. The substance or parietes of the bowels may be considered as affected in these cases, even although the external surface may present no further lesion than red vessels shooting into it. Occasionally, in addition to this state, the red capillaries in the inflamed peritoneal coat are connected with the effusion of coagulable lymph, particularly in the parts where they are most numerous, the lymph or albuminous exudation existing in specks, or in considerable spots or patches, on the serous surface. When, however, these latter appearances are remarked, the interior of the inflamed intestine frequently presents more serious changes than yet noticed. The villous surface is then deeply inflamed, and seems abraded or excoriated in parts. It is sometimes, in other parts, covered by patches of lymph, or of an albumino-puriform, or muco-puriform fluid, or by a sero-sanious matter; and it is often also ecchymosed in numerous points or specks, or it presents still larger marks of sanguineous infiltration. In other cases, portions of a dark, slate-coloured, or sphacelated hue are observed, with or without ulcerated specks, or even large ulcers, which have nearly penetrated as far as the external coat, in adjoining parts." Sometimes then, ulcers actually perforate the intestinal wall, and give rise to escape of the contents. "In the forms of enteritis in which the substance of the intestine, or its peritoneal coat, is chiefly affected, either primarily or consecutively, the whole of the coats are often very vascular, red, or of a brick-red colour, and are readily torn." Suppuration sometimes takes place between the coats, as in an interesting case recorded in the Pathological Society's Reports, 1851-52, by Dr. Hare. General peritonitis not unfrequently occurs, in which the omentum may markedly participate, becoming greatly thickened and red.

Croupous and Diphtheritic Inflammation.—The intestinal mucous surface is sometimes the seat of a kind of inflammatory process which is mostly subacute or chronic, and gives rise to an exudation much resembling that of croup. It is remarkable that the attacks often recur several times, each presenting a stage of irritation, which ends in the formation and throwing off of the false membrane, after which there is a pause. The quantity of the exudation varies much in different cases: sometimes it forms a layer of some thickness, extending pretty uniformly over the surface, or appearing in the stools as tubular casts of the intestines; sometimes it is as thin as a wafer, or consists merely of tattered shreds. In one case mentioned by Dr. Copland there were also shreds of dysmenorrhœal false membrane discharged from the uterus, but not at the same time as those from the bowels. Rokitansky mentions the occurrence of less consistent fibrinous exudations, which probably approach more or less closely to those of diphtheritis. They are, as he observes, "the expression of a constitutional affection," the results of an altered blood crasis. Such affections are commoner in children than in adults, but are always rare. False membranes, similar in appearance, are sometimes formed in dysentery a disease, which will be spoken of hereafter. The appearances just described are not known to occur in cases of ordinary diphtheria (of the throat), but swelling of the solitary and Peyer's glands in the ileum has been observed in that disease.

INTESTINAL AFFECTION IN ENTERIC FEVER.

The changes which take place in the intestinal mucous membrane in continued fevers have been most diligently examined, and minutely described by Rokitansky and others, but our limits forbid us to do more than give a short and comprehensive account of the series of morbid changes occurring in typhoid or enteric fever. We observe the following series of changes:—Hyperæmia to a greater or less extent is set up around the solitary follicles, and in and around Peyer's patches. Enlargement and distension of these glandular structures proceed nearly *pari passu* with the hyperæmic congestion. After a certain time, the length of which varies in different cases, the contents of the enlarged glandular masses soften, break down, and are discharged. The cavity which remains on the mucous surface, constitutes the typhoid ulcer, to which Rokitansky attributes the following character:—(1) Its form is elliptical, round, or irregular, and sinuous, according to the shape of the part which has been affected. Thus, a large patch, when destroyed, gives rise to an elliptical ulcer; a smaller or solitary gland to a round one; partial destruction of a patch will produce an irregular ulcer. (2) The size of the ulcer varies from that of a hemp-seed to that of a half-crown. (3) Those of an elliptic shape are always situated opposite to the insertion of the mesentery, and have their long axis parallel to that of the intestine. The typhoid ulcer very rarely indeed forms a zone. (4) "The margin of the ulcer is invariably formed by a well-defined fringe of mucous membrane, which is a line or more wide, detached, freely moveable, of a blueish red, and subsequently of a slaty or blackish-blue colour. (5) The base of the ulcer is formed by a delicate layer of submucous tissue which covers the muscular coat; like the marginal substance, it is quite void of morbid growth. (6) The small intestine is the seat of the ulcerative process, and the lower third is most liable to be involved—the number and size of the ulcers increase as they advance towards the ileo-cæcal valve.”

Fig. 135.



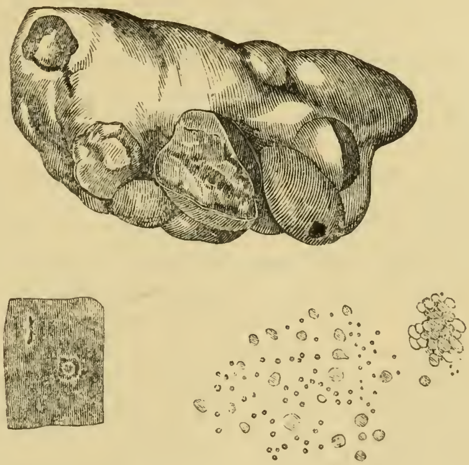
Typhoid ulcers in small intestine. Death from hæmorrhage. The outline figures represent vertical sections. In the upper figure the margins of the ulcer are thickened; in the lower they are clean cut, as if punched.

We must add with respect to the last character, that the ulcerative process is by no means confined to the small intestine; we have seen the mucous membrane of the large intestine, down to the rectum, riddled with ulcers. They were many of them of large size, and had clean cut, non-thickened margins. This condition, indicating the absence of reparative action, is not nearly so frequent as that of thickening and induration, which generally take place to some extent in the side of the ulcer. The bottom of the ulcer is commonly formed by the submucous tissue; sometimes the muscular fibres are completely exposed. This, however, is generally the result of secondary advance, subsequent to the expulsion of the morbid products. Rokitansky particularly insists on the point, and we think he is correct, that when an ulcer increases in depth so as to perforate the intestine, it advances not by continued deposition and softening of the same matter as that originally produced, but by simple extension of the ulcerating action.

The matter which causes the tumefaction of the agminate and solitary glands chiefly consists of actual multiplication of the corpuscles in the (agminated lymph and solitary) glands accompanied by a certain amount of exudation; these newly-formed elements seem to be particularly liable to decay, so that a large portion of the mass may be found in the condition of granular degeneration, or converted into amorphous molecular detritus. Black granules and grains of pigmentary matter are often present in it, but they are by no means peculiar to the typhoid state. They give to the glands a black dotted appearance, as seen by the naked eye, and this we have observed more than once after death from other causes than fever.

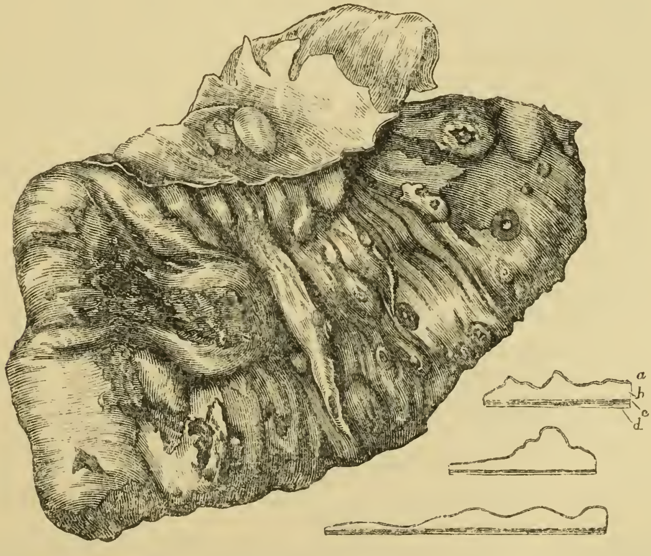
Lymphatic Glands.—The mesenteric glands become invariably enlarged in all cases of intestinal affection, just in the same way as an inguinal gland enlarges when there is a chancre on the glans penis. Their enlargement seems to be simply the result of irrita-

Fig. 136.



Inflamed mesenteric glands in typhoid and so-called typhus matter. At the lower and left part is represented a small ulcer in the mucous membrane, in which ulceration is seen extending round the central deposit, which is still *in situ.*

Fig. 137.



Typhoid ulcers in various stages. The outline figures are vertical sections which show the elevation of the mucous membrane by submucous deposit.
a, Mucous membrane. *b*, Submucous tissue. *c*, Muscular coat. *d*, Peritoneal coat.

tion; we have found nothing in their substance besides the normal nuclei but granular and amorphous matter, and some celloid corpuscles or cells. The vessels of their capsule are generally much congested, as well as those which penetrate their interior. Rokitansky states that "the mesenteric glands decrease in size, as soon as the detachment of the intestinal morbid growth has commenced." Of course they must remain for some time more congested with blood, and larger than natural, even under the most favourable circumstances.

Resolution and Healing.—It by no means necessarily follows that ulceration takes place after a patch or a single gland has been enlarged; the exudation may liquefy and be again absorbed into the blood, and the part return to its normal condition. Cicatrization of the ulcers is not unfrequent, as Sir Thomas Watson testifies. He says, "The ulcerated surface seems to clothe itself afresh, by degrees, with a new mucous membrane, which is thin, however, and adherent to the subjacent tissues, and does not slide over them when pressed between the finger and the thumb, as the healthy portions of the coats of the bowel will do upon each other. And in the place of the cicatrix there is usually to be seen a manifest puckering, and a number of little wrinkles or lines, radiating from a common centre."

Rokitansky speaks of the new-formed membrane as a serous lamina, which becomes at its circumference as it were dovetailed in between the muscular and mucous coats. He confirms the observation of Sebastian, that small villi sometimes form upon this lamina, even before its union with the mucous membrane. In most instances, however, the absence of villi forms one of the distinctive features of a cicatrix. We feel much hesitation in accepting Rokitansky's absolute assertion, that the cicatrix of a typhus ulcer never in any way gives rise to a diminution of the calibre of the intestine. Dr. Carswell speaks positively of the occurrence of fatal ileus in persons who had suffered some months before from typhoid fever, the cicatrix of an ulcer being found after death, which had destroyed the muscular coat around the whole circumference of the tube.

Secondary Ulceration.—Instead of cicatrization, or even when partial healing has taken place, the ulceration sometimes extends in a remarkable and irregular manner from its original seat, by a simpler process of destruction than that by which the typhoid ulcers were originally formed; and this secondary ulceration has even been compared to gangrene. The ulcers thus produced, which might be called "post-typhoid" ulcers, are quite irregular in shape, and have not the typical characters of typhoid ulcers given above. Ulcers of the colon met with in typhoid fever appear to be often of this kind.

Consequences of Typhoid Ulcers.—Perforation and hæmorrhage are the two most formidable results of typhoid ulcers. The former may take place at any period of the disease, but rarely happens before the complete separation of the slough, since it depends on an extension of the morbid process to deeper parts than its original seat. According to Dr. Murchison, perforation may occur in three ways, either:—(1) by rupture of the attenuated coats; (2) by continuance of the ulcerative action leading to a pin-hole perforation; (3) by sloughing of the entire thickness of the bowel and dropping out of the sloughs. Trousseau declares that "most commonly, the blood is exhaled by the mucous surface, just as in epistaxis" (vol. i. p. 230). Hæmorrhage may occur apparently from mere excessive hyperæmia in early stages, or from the margin of the ulcers, but the most severe bleedings occur when the progressive ulceration has reached some comparatively large vessel, which may sometimes be seen distinctly eroded on the floor of the ulcer.

TUBERCULAR DISEASE OF THE INTESTINES.

Tubercle selects the small and large intestines not unfrequently as its seat of deposit. It is met with more frequently in children than in adults, in the proportion of sixty-one to forty-three, and is more than twice as frequent in the small as in the large intestines. In the majority of cases the affection of the intestines is secondary to that of the lungs, and usually takes place after the tubercles in the latter have begun to suppurate, and the cachexia has become fully developed. The course of intestinal tuberculosis is "frequently chronic, but much often acute." The seat of the deposit is the submucous tissue or the corium of the mucous membrane, it is certainly subjacent to the basement membrane, and not contained in the follicles, as Dr. Carswell taught. Rokitansky states that there is, in the chronic form, "no perceptible inflammatory action, and the disease appears in the shape of the grey transparent granulation, which softens at its centre, and is gradually converted from within outwards, into the yellow cheesy tubercle. It seems blended with the mucous membrane, and projects into the intestinal cavity in the shape of a sessile, hard nodule. Considerable inflammatory action attends upon acute tuberculosis. The disease affects first Peyer's patches, then the solitary glands, and lastly, other parts of the mucous tissue." The affection of the Peyer's patches is often partial, some follicles escaping altogether, while adjacent ones are in an advanced stage of disease. The affected follicles enlarge and become infiltrated with small cells, which are also found in the surrounding parts. Each follicle is at first grey and translucent, then a yellow opaque speck appears in the centre, the caseous change spreads through the mass, and at length softening commences, which spreads from the surface, so that in Peyer's glands several small ulcers are formed, while the solitary glands are destroyed and converted each into one ulcer. The walls being infiltrated with small cells, which are often collected into roundish granulations, or tubercles, pass through the same series of changes, and ulceration thus extends in an irregular manner beyond the original follicles, till the form of the original structure is quite lost. It may happen that the affected part is so completely destroyed that no intermediate stages are seen, and then (as in tubercular ulcers of the larynx) it is difficult to distinguish them from simple ulcers.

The surrounding tissue is hyperæmic, red, and swollen, and the margin of the ulcer firmly attached, rounded and indurated; its base, usually formed by condensed areolar tissue, may contain or not tuberculous matter. The enlargement of the ulcer in depth and in extent is thus effected by a process of essentially the same kind as is observed in cavities of the lung. The margin and the base become infiltrated with tuberculous products which soften and decay, and so the destructive process continually advances. It thus penetrates the muscular coat which becomes studded with granulations, formed, according to Klebs and others, in the lymphatic vessels, and finally may reach the serous covering, and produce a crop of granulations on the peritoneal surface. This may be the starting point of general tubercular peritonitis, but the peritoneal tubercle is, on the other hand, very often primary. Perforation of the intestinal wall sometimes occurs, the ulcer retaining throughout its original character, in which respect it differs from the typhoid. The fatal event is often prevented by timely exudation of fibrine on the serous surface, which either thickens the wall or else unites it to an opposing surface. Mr. Ancell contests the statement that the tuberculous ulcer always retains its original character, he does not believe that ulcerations, the result of tuberculosis, are always produced by the deposit of tubercle. He views them as the result of "the ulcerous diathesis of the disease." We have noticed above the only ground which exists for such an opinion.

The mesenteric glands are enlarged by the special deposit, and often to considerable size. This, however, it may be stated, is not the essential circumstance in the disease formerly called *tabes mesenterica.*

Tuberculous ulcers heal by a cessation of the morbid deposit taking place, and subsequent effusion of plastic fluid, which becomes organized into the cicatrix tissue before described. The contraction of this, if the ulcer has been of a large size, or extended round the intestine, may cause more or less contraction of its canal. Tuberculous disease generally, but not always, selects the lower part of the ileum as the seat of its chief ravages; it oftener descends, we think, to the cæcum and colon than extends in the upward direction.

Intestinal tubercle is nearly always secondary to that of the lungs, and it has been suggested by Klebs that the intestinal disease is produced by swallowing morbid products derived from phthisical lungs, a hypothesis also supported by the fact that tubercle in the intestines tends so strongly to spread downwards. Peritoneal tubercle seems to have but little tendency to produce the same disease on the mucous surface.

Distinction between Tubercular and Typhoid Intestinal Ulcers.—Since these are both found in the same parts of the intestine, and have many points of similarity, it is important to be able to distinguish them, but sometimes difficult. The chief points to be regarded are the following:—(1) *Form.* Typhoid ulcers are more often oval, with the long axis in the direction of the bowel, corresponding to the shape of the Peyer's patches, and even when they start from the solitary glands, extend down rather than round the intestine. They do not often extend beyond the boundary of a Peyer's patch. These characters, however, only hold good in comparatively recent ulcers; when they have existed a long time (two months or more from the commencement of the fever) their shape may become very irregular. Tubercular ulcers, on the other hand, seldom correspond in outline to the Peyer's patches, and, if oval, the long axis of the ellipse more often goes round the intestine than down it. They often extend beyond the margin of the patch, even without involving the whole of it. When starting from the solitary glands they extend in a quite irregular manner. Their extension seems in fact to be determined rather by the course of the blood vessels, than by the glandular structures, and, as the former encircle the intestine, the ulcers not unfrequently form a complete ring. (2) *Situation.* The typhoid ulcers are always at first situated on the side remote from the mesenteric attachment since the Peyer's and other glands are found there; tubercular ulcers may occur in other parts, even at the mesenteric attachment. (3) *Colour.* In the typhoid process, before ulceration is complete, the necrotic sloughing mass is found stained yellow by fæces, or with fæcal matter adhering to it, which is not the case with tubercular ulcers. (4) *Margin.* The margins of both are raised, but in the typhoid ulcers, simply swollen and injected; in the tubercular, infiltrated with tubercular or caseous matter, while there are often distinct outlying tubercles. In typhoid the wall is very abrupt or even somewhat overhanging, as may be seen by directing a gentle stream of water upon the ulcer. In tubercular ulcers the descent is gradual or by successive steps. (5) *Surrounding parts.* Actual tubercles are sometimes found in the mucous membrane surrounding tubercular ulcers, or their floor, or on the serous covering of the intestine, but they are not constant. They are of course wanting in typhoid ulcers, and the peritoneal covering over these is usually natural. (6) *Cicatrization.* Tubercular ulcers form a more decidedly fibrous cicatrix, and more often lead to contraction of the intestine than typhoid. In fact, it is doubtful whether the latter ever do so.

These distinctions will generally be sufficient, though cases of difficulty will arise, especially when typhoid ulcers have lasted a long time. The state of the mesenteric glands and of other organs, must then be taken into account.

INTESTINAL CHANGES IN DYSENTERY.

The morbid changes in dysentery have their especial seat in the large intestine, the ileum being sometimes (Dr. Copland says very often) involved, but always in a less degree. Our observation quite accords with Rokitansky's, that, "as a rule its intensity increases from the cæcal valve downwards," so that the sigmoid flexure and the rectum are found most severely affected. It commonly runs, Rokitansky says, an acute course, "though it is frequently chronic in the milder degrees; this, however, does not materially alter its character." Dysentery presents itself to the medical observer under a very great variety of forms, but it would be impossible to range the *post-mortem* appearances in corresponding groups. All that can be said in general is, that both the symptoms during life and the textural lesions, will coincide in indicating whether a given case is to be considered of sthenic or asthenic character. Looking then upon dysentery generally as inflammation, more or less acute, of the mucous membrane of the large intestine, and premising that it is very prone to pass on into a lingering chronic state, we shall follow Rokitansky's account of the changes produced by the disease. He considers them "as divisible into four natural degrees."

In the lowest the mucous membrane in its projecting folds is injected, swollen, and softened, its surface seems excoriated, the epithelium is detached as a greyish-white layer, or elevated by effusion into small vesicles containing serum, as in a case we witnessed, or it may be mingled with amorphous matter in an exudation of dirty grey and reddish colour covering the surface. Dr. Baly, who mentions the detachment of the epithelium and its mingling with amorphous matter, describes the solitary glands in the first stage as being enlarged, forming round prominences, which in a chronic state, by sloughing and ulceration, assume the form of open sacs. The mucous and submucous coats become thickened.

"In the second degree the textural alterations are not limited" to the projecting folds, "but extend over a larger surface, still, however, presenting a greater development at one part than at another. The mucous membrane is invested to the same extent, by a dirty grey layer, consisting of desquamated epithelium and a thick glutinous exudation; or this may already have been removed, and the subjacent mucous membrane, in either case, appears converted into a soft, sanguineous, pale red and yellowish gelatinous substance, which may be easily detached." The submucous tissue becoming infiltrated, gives rise to more or less numerous protuberances, on the internal surface of the intestines; these "correspond to those points of the mucous membrane, at which the morbid affection is most developed," while in the intervening portion there is not much change beyond slight redness and swelling. The intestinal cavity is dilated by the pressure of exhaled gas upon the semi-paralyzed muscular coat, and contains a mixture of effused lymph and blood, together with mucous liquid and fæces. Dr. Baly marks his second degree by the sloughing of the solitary glands, either principally or equally with the surrounding mucous membrane. In this way are formed either clear circular ulcers of various depth, or large excavations. The prominent rugæ are chiefly affected. Rokitansky also remarks, that the affection of the follicles may predominate, and states that the anatomical condition is the same as that already described in connection with catarrhal inflammation as attendant upon lientery.

"In the third stage the protuberances are set more closely together, the mucous membrane investing them partly retains the same condition as in the former, partly" is converted into a slough, which is here and there blended with desquamated epithelium "and the exudation, and is firmly attached to them; it is of a dark-red, or blackish-brown, or greyish-green colour." In some cases the infiltrated and thickened submucous tissue is in great part exposed, being covered here and there by the remnants of the mucous membrane, "in the shape of solitary, dark-red, flaccid, and bleeding vascular tufts, or as dilated follicles, which are easily removed." The protuberances are occasionally found to have coalesced, and the intestine then presents an uneven plicated surface, accompanied by an equal degree of infiltration and thickening of its parietes; the mucous membrane is uniformly affected over a large extent, and there are no free interstices. The contents of the intestine are of a dirty brown or reddish ichorous, fœtid, flocculent, and grumous character.

In the fourth and highest degree, the mucous membrane degenerates into a black, friable, carbonified mass, which may often be subsequently voided in the shape of tubular laminæ (so-called mortification of the mucous membrane). The submucous cellular tissue appears to be previously infiltrated with venous blood, or a sero-sanguinolent fluid; or it is pallid, and the blood contained in its vessels is converted into a black, solid or pulverulent mass; subsequently it shows purulent infiltration, in consequence of the reactive inflammation which is induced in the lower, healthy strata, for the purpose of eliminating the gangrenous portions. Dr. Baly's third degree seems to correspond to the fourth of Rokitansky, he describes the mucous membrane as converted into a gangrenous slough, glands and all alike, the blood being dark and coagulated in the submucous tissue. The prominent rugæ are first and most severely affected, the intervening portions being swollen and red. All the coats sometimes are much softened, and the submucous tissue becomes sloughy. The changes just enumerated as occurring in the most extreme degree of the disease, are of much the same kind as those which Dr. Copland describes to take place "in the most malignant varieties, and in the scorbutic complication. The internal surface of the whole digestive tube is," in these cases, "of a livid purple, or dark colour, with patches of ecchymoses, excoriation, ulceration, and sphacelation. The villous coat, particularly in the seat of ecchymoses, may readily be rubbed off; and the ulcers have a foul and dark appearance. The liver is sometimes large, soft, and spongy; at others, pale and soft, especially in cases where the loss of blood has been very large. The spleen is sometimes so softened as to appear semi-fluid or sphacelated. The heart is often partially softened or flaccid; the pericardium and pleural cavities containing a bloody, dark, and dirty serum. The lungs are often congested; the bronchial lining dark or ecchymosed; and the blood, in all the large vessels, is semi-fluid, black, and of a very loose texture." "In prolonged inflammatory cases, thickening and almost cartilaginous induration of a considerable part of the colon are not unfrequent, the thickened or indurated portion being also contracted in calibre. In such cases, the parts above the contraction are greatly distended, the coats being thinned, ulcerated, and even lacerated;" so as to give rise to effusion and fatal peritonitis. The darkly-congested mucous membrane is often discernible through the peritoneal and other coats, especially if, as is often the case, the intestine is distended. In the severer cases the serous membrane is dulled and discoloured, and sometimes covered with a brownish ichorous exudation. The mesocolic lymphatic glands are swollen and congested. In the dysentery which occurs in this country, abscess of the liver is rare; a case, however, is recorded in the Report of the Pathological Society, 1851-52. Dr. Baly has never met with it during his experience at the Millbank Penitentiary, where the disease is very common; but in India it is said to occur in nearly half the cases. We have recorded, in the Report of the Pathological Society, 1847-48, an instance in which the destruction of the mucous membrane was confined to the interstices of numerous prominences or ridges, these being themselves the sole remnants of the mucous tissue. This is the converse of the more usual condition, in which the prominent parts are most affected. After extensive ulceration has occurred, reparation may be effected in the usual way by organization of plastic exudation into a smooth fibroid layer which constitutes a cicatrix. This probably may be covered after a time by an epithelial layer; but it has not been shown yet that the follicles of Lieberkuehn are reproduced. When extensive destruction of substance has taken place, the cicatrix tissue "is frequently condensed into fibrous bands, which form corded projections into the intestinal cavity, interlace with one another, and not unfrequently encroach upon the calibre of the intestine in the shape of valvular or annular folds, thus giving rise to a stricture in the colon of a very peculiar form." Instead of reparation taking place, the disease may continue in a chronic though altered form; the remaining mucous membrane being in a state of chronic catarrhal inflammation, and the intervening parts being the seat of suppuration with formation of sinuses and abscesses.

Dysentery, as described above, is undoubtedly a specific disease; probably in most cases, some persons would say in all, communicated by infection. It has many points of resemblance to diphtheria, so that some have spoken of it as intestinal diphtheria.

INTESTINAL CHANGES IN SPECIFIC DISEASES.

Intestinal Changes in Scarlatina.—The agminated and solitary glands of the ileum are found in some cases of scarlet fever enlarged and vascular, as in early stages of the typhoid change. More rarely the process goes on to actual ulceration. It is probable that some degree of the same change may accompany other acute febrile diseases. It has been several times observed in diphtheria.*

Dr. John Harley† has made an extensive series of observations on the intestinal changes in scarlet fever. He has found in twenty-seven cases out of twenty-eight the Peyer's patches, especially in the lower third of the ileum, elevated and injected, sometimes swollen like a cock's-comb, or of a dark purple colour, and occasionally almost ecchymotic. The solitary glands of the ileum were, in half the cases, enlarged, granular, and whitish, like sago-grains. In a smaller number of cases the solitary glands of the colon were similarly enlarged, and more rarely the general mucous surface was injected. In one case acute desquamation of nearly the whole of the mucous membrane of the large intestine was observed. He never saw ulceration of the solitary or agminated glands, except in some cases which he regarded as typhoid fever supervening on scarlatina.

Simple or unexplained Ulceration of the Intestines.—Cases sometimes occur in which ulcers of the intestines, not unlike those associated with enteric fever, are met with, but where there is no evidence of this disease having existed, nor of any other associated morbid lesions. This may be the case in the ileum or in the colon. Some of these puzzling cases may be found recorded in "Trans. Path. Soc.," vol. xviii. pp. 101-104, by Dr. Whipham and Dr. Dickinson; who observed numerous ulcers in the colon, for which no cause except chronic constipation could be assigned.

Intestinal Changes in Cholera.—The appearances in this disease are variable, and in rapid cases, dying in the stage of collapse, very slight changes only are sometimes seen. The mucous membrane is injected, sometimes very brightly, and œdematous. The solitary follicles and also the Peyer's patches, especially the former, are often enlarged, of a whitish colour, and firmer consistence than in simple catarrhal conditions. Occasionally ulceration occurs and small follicular ulcers are formed, while the process may, in the colon, extend so as to form wide shallow ulcers (Klebs). Patches of false membrane, resembling those of diphtheria, are sometimes met with in later stages; they are described as firmly adherent and not removed without some lesion of the mucous membrane.

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* Cayley, "Trans. Path. Soc.," vol. xvii. p. 123.

† "Med-Chir. Trans.," vol. lv. p. 103.

The contents of the intestines after death from cholera have been very carefully examined. They are chiefly made up of a large quantity of very watery fluid, holding in suspension flakes of mucus, large quantities of detached epithelium in the form both of separate cells and continuous layers (chiefly in small intestine); obscure, finely-divided granular matter: and certain peculiar organized bodies, regarded as vegetable parasites. This agrees with the composition of the well-known "rice-water" stools passed during life; except that the latter contain no epithelium, which is therefore probably detached by *post-mortem* maceration. The liquid portion, as has been pointed out by Klebs, agrees very closely in the composition with the secretion poured out in the ileum after the section of the intestinal nerves (by Moreau), containing about eleven to fourteen per mill. of solid parts, of which three to six are organic and eight inorganic. It contains in solution chiefly chloride of sodium with little potash or phosphoric acid and usually urea. The organized bodies found both in the intestinal contents and the stools appear to belong to a species of fungus; sometimes thought to be the actual cause of the disease.

Cholera Fungus.—Many observers, of whom the earliest were Budd, Brittan and Swayne, in 1849, the latest Beale, Klob, Hallier and Thomé, have observed some form of fungus in choleraic stools. The forms usually seen are those of cysts or sporangia, containing spores which ultimately escape by rupture of the envelope. The cysts are found detached or connected in rows. According to Hallier, a form of jointed fungus may be obtained by cultivation from choleraic stools which produces sporangia precisely similar to these; and from the spores the fungus itself may be reproduced. He compares the fungus to certain forms parasitic upon grasses; and as it is different from any European form, he suggests that it may be derived from an Asiatic form parasitic on rice.

Beside the special forms of parasite above described, ordinary bacteria (or micrococci) and sarcinæ are found in the evacuations, but there is no reason for ascribing to them any special relation to the disease.

Intestinal Changes in Leuchæmia.—The follicles of the intestinal tract show their connection with the lymphatic system by sometimes participating in the changes which lymphatic structures undergo in this disease (p. 73). The change consists, usually, merely in enlargement, by multiplication of the lymphatic corpuscles, but distinguished (according to Klebs) from simple enlargements by their softer consistency and greyish colour. The cell-production often appears to extend beyond the boundaries of the original follicle, and, in a few cases, as in one figured by Virchow (Krankh. Geschwülste, II. 569), actual ulceration has been seen. In this case, cellular infiltration extended through the walls of the intestine almost to the serous covering, and produced flat varied patches on the mucous surface, which were in great part ulcerated, so that they much resembled tubercular ulcers. There were, besides, the usual leuchæmic swelling of the spleen, and lymphatic masses in the liver and kidneys. Similar ulcers have been seen in the duodenum.

It is interesting that very similar products have been observed in the intestine in a case of general lymphatic enlargements and growths, (constituting the disease known as Adénie, Pseudoleukæmia, or Hodgkin's disease,) unaccompanied by increase of white corpuscles.* Lymphatic new formations were found in the liver, kidneys, heart, and other organs, as well as in the duodenum, where the growth formed a mass more than an inch and a quarter in thickness, chiefly in the mucous coat, but partly proceeding from the cytogenous or adenoid tissue lying under the basement membrane. Smaller masses occurred at intervals, both in the small and large intestine, and were, in all cases, composed of lymphatic corpuscles, held together by a stroma.

Syphilitic Affections of the Intestinal Canal.—Syphilis very rarely affects any part of the intestines except the rectum, where two forms of disease are met with, viz., ulceration and stricture. Syphilitic ulcerations much resemble, according to Virchow, dysenteric ulcers, but are distinguished partly by their more uniform and level ulceration, partly by their situation, which is near the anus, and not in the upper part of the rectum or sigmoid flexure. He thinks it uncertain whether they begin with condylomatous or gummatous growths, or whether they are, as some think, local, that is, primary affections. The fibrous scars of these ulcers give rise to formidable strictures of the rectum.

Ulcers of other parts of the intestine have been found in newborn children affected with hereditary syphilis, and, in a few cases, in adults. Klebs describes a case of an undoubtedly syphilitic man, in whom were found some considerable ulcers near the ileocæcal valve and lower part of ileum, almost surrounding the intestine in a ring; while numerous small ulcers existed in other parts. They had a firm fibrous base, and the edges were beset with villous projections.

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* Murchison, "Trans. Path. Soc.," vol. xx. p. 198.

Fatty tumours, sessile or pediculated, occur in the intestinal canal; they originate in the submucous tissue, where fat vesicles always exist, and grow inwards. *Serous* and *fibro-serous cysts* are met with but very rarely between the intestinal coats. *Fibroid nodules*, not exceeding the magnitude of a pea, are sometimes found in the submucous tissue. In a case recorded by Dr. Duckworth, four fibrous tumours, from the size of a bean to that of a walnut, were found in a dilated portion of the ileum. ("Trans. Path. Soc.," vol. xvii. p. 125.) *Calcareous concretions*, formed by the deposition of earthy matter in new-formed fibroid tissue, in obsolete tubercle, or desiccated pus, or fibrinous exudation, occur very rarely. Rokitansky describes *erectile growths* to exist in the intestinal canal, either in the form of sessile tumours or pediculated polypi. We are not sure whether he would include under this head instances of fibrous polypi, such as two recorded by Mr. P. Hewett, in the Report of the Pathological Society for 1846-47, which, though of marked fibrous structure, were livid in appearance, and pretty plentifully supplied with blood-vessels. The presence of these polypi may give rise to invagination and its consequences.

Cancer not uncommonly attacks the intestinal canal; it is far more frequent in the large than in the small intestines; out of 378 fatal cases from this cause, in 221 the disease was located in the rectum. It is almost always primary, but sometimes arises by extension from neighbouring organs; and very rarely secondary growths occur in the mucous membrane, or in the Peyer's glands. Dr. Walshe seems to consider the small intestines to be more frequently affected than Rokitansky does; the latter says that "the colon is almost exclusively the seat of cancerous degeneration," while "the small intestine is scarcely ever the primary seat of cancer, except in the case of acute and extensive encephaloid disease." In the Report of the Pathological Society for 1847-48, there is recorded one case of cancer (colloid) of the small intestines and mesentery, and four of the cæcum, colon, and rectum. The duodenum and upper part of the jejunum are the parts of the small intestine most frequently affected, the rectum and sigmoid flexure those of the large which most frequently suffer. Mr. Curling has met with an instance of epithelial cancer in the coats of the intestine. When the disease has its seat in the rectum, it most usually occurs at from two to three inches above the anus, according to Dr. Walshe, and tends to spread upwards rather than downwards.

Cancer of the intestines appears to occur in the same forms as in the stomach, viz., scirrhus, medullary, colloid, cylinder-celled epithelioma; and in the rectum, flat-celled epithelioma. The cancers described as medullary commence in the submucous tissue, and often grow in a ring all round the bowel, which then usually becomes constricted, though, occasionally, on the other hand, dilatation takes place. Ulceration is produced on the mucous surface, and sometimes the growth projects into the cavity of the intestine. Extending also outwards, it infiltrates the whole wall and penetrates to the peritoneum, where large masses are sometimes formed; so that it may be difficult to say whether the growth originated in the bowel or the peritoneum. Adhesions to neighbouring parts are also formed. Under the name medullary have, doubtless, been included many tumours which would now be called round-celled sarcoma, or lympho-sarcoma. This form of cancer is found most frequently in the rectum, next in the sigmoid flexure or other parts of the colon; very rarely in the small intestine. Scirrhus is far commoner in the rectum than the form just described, and is only occasionally found in other parts. Its course is generally the same as that of medullary, except that it forms a firm fibrous stroma, which always causes great contraction of the bowel, and that there is beside much fibrous or muscular hypertrophy in surrounding parts; so that scirrhus of the rectum may come to have a striking resemblance to scirrhus of the pylorus.

Colloid cancer is more common in the intestines and peritoneum than anywhere else in the body. It forms flat masses, or, sometimes, complete rings, causing obstruction. In its manner of growth and consequences it agrees generally with medullary and scirrhus. According to Klebs, it is most often found at the hepatic flexure of the colon, and at the ileo-cæcal valve.

Cylinder-celled epithelioma is, perhaps, the commonest form of cancer in all parts of the intestine, and is very nearly allied to simple glandular tumour or adenoma. This forms tumours of moderate hardness, sometimes white, opaque, and medullary, sometimes translucent, so as even to approach colloid in appearance. It causes great contraction of the intestine. On microscopical examination this form of cancer is found to be made up chiefly of cylindrical epithelium, arranged much in the same manner as in intestinal glands. We have seen it in the duodenum and in the colon. A remarkable case from the duodenum is described by Mr. Coupland ("Trans. Path. Soc.," vol. xxiv. p. 103). Secondary tumours sometimes occur in the liver and lungs, which resemble the primary in structure. Some cases of this have been described as medullary.

Flat or pavement-celled epithelioma is only found in the rectum near the anus.

An ossifying cancer from the rectum is described by Mr. Wagstaffe ("Trans. Path. Soc.," vol. xx. p. 176). It appeared to be an ossifying scirrhus, but the case is of extraordinary rarity.

Consequences of Intestinal Cancer.—When any cancerous growth assumes an annular form encircling the intestine, it produces gradually increasing constriction, which may advance to such an extent that the canal is reduced to the diameter of a goose or even a crow-quill. A tuberiform growth, occupying only one side of the intestine, also gives rise to considerable constriction; but this is not so great generally as in the former case. The narrowing of the passage, it is manifest, will be greatest while the growth remains in its original (crude) state; but if, as not unfrequently happens, sloughing and ulceration take place in the morbid mass, the obstructed passage will be again more or less reopened. In many cases, however, the obstruction to the passage of the contents is such that the portion of the canal above the structure becomes immensely dilated, with its muscular tunic much hypertrophied, and its mucous on the contrary sometimes thinned, while that below contracts upon itself and becomes very small. Death often takes place in cases of intestinal cancer from the supervention of ileus and inflammation. The ileus is set up not only in consequence of the stricture preventing the passage of the contents of the bowel, for it may ensue when the canal is still tolerably pervious; but from the paralyzed condition of the walls of the intestine in the dilated and distended part, and from the masses of the fæcal matter which accumulate there. Inflammation attacks first the dilated part of intestine, "and is there most intense. This portion is discoloured, of a dark blue or reddish aspect, its coats are infiltrated with blood; the serous lining covered with exudation is easily detached, the muscular coat is discoloured and friable; the mucous membrane, owing to its distension, is devoid of plicæ, villi, or follicles; dark red, distended at some parts with coagula, and sloughy." There is usually ulceration of a portion of bowel thus distended; sometimes perforation and its consequences take place.

MORBID CONDITIONS OF PARTICULAR PARTS OF THE INTESTINES.

Typhlitis, Perityphlitis.—The frequent occurrence of catarrhal inflammation in the cæcum has been already mentioned; but Rokitansky directs attention to a particular form which he terms *Typhlitis stercoralis*, indicating thereby its production by accumulation of indigestible and fæcal matters in this situation. Sedentary habits and "rheumatism" of the muscular coat of the bowels are also stated to be causes of the disease. We believe the proximate cause to be atony of the contractile fibres. "Removal of the accumulated fæces, and avoidance of fresh accumulations, generally suffice to establish a cure. If this is not effected, ulcerative destruction of the mucous membrane, and continued sinuous suppuration of the muscular coat, result. In this manner rapid perforation of the intestinal parietes, and especially of the posterior side, may follow, either inducing extensive inflammation, ichorous destruction of the cellular tissue in the iliac and lumbar regions, and death; or giving rise to general peritonitis" from transmission of the inflammation to the serous membrane.

Inflammation sometimes attacks the lax cellular tissue which lies between the posterior surface of the cæcum and the iliac fascia, and is apt to pass into suppuration, a condition which has been called *peri-typhlitis.* In many instances it is doubtless set up by pre-existing, perhaps chronic, inflammatory disease of the bowels; but in others it occurs idiopathically, or metastatically as Rokitansky states. A calculus descending along the ureter has given rise to abscess in this situation. The purulent matter diffuses itself often for some considerable distance beneath the serous membrane; it has been known to make its way up as high as the kidney, and as low as the interspace beneath the rectum and bladder. The abscess often opens into the cæcum, and also externally. When the catarrhal affection of the cæcum exists in a chronic form, it may cause the condensation of the surrounding cellular tissue, and shrivelling of the intestine itself, so that it "is found converted into a slate-coloured capsule, with dense parietes, the size of a walnut or of a pigeon's egg."

The cavity of the *vermif. appendix* often affords lodgment to indurated pellets of fæcal matter, cherry-stones, or other such bodies, which cause irritation and inflammation, thickening of its coats, and subsequent ulceration. If the irritating substance can be got rid of, and the ulceration ceases, the appendix shrivels up entirely, or in part, according to the extent of the mischief, assuming a lead or slate colour. In two very interesting cases recorded together in the Report of the Pathological Society for 1847-48, the vermiform appendix protruded as a hernia, and had undergone ulceration in this situation. In the first, after an abscess and sinuses had been laid open, a small piece of bone, of triangular shape, and with sharp angles was discharged, and recovery very quickly took place. Death occurring from a different cause not long after, it was found that the vermiform appendix was lying in the inguinal canal, it was enlarged to three times its usual size, its coats much thickened, and its apex opaque, contracted, and adherent to the bottom of the canal. In the second case, there was a swelling of the scrotum caused by the hernial protrusion of omentum enveloping the vermiform appendix. Abscesses and sinuses formed in the part, healthy pus was at first discharged, afterwards sanious and offensive matter; the quantity of discharge was profuse, but varied in quantity, and was frequently of a pale orange colour. Death occurred from exhaustion, and it was found that the appendix, healthy in structure all the way down to near its blind extremity, was ulcerated at about half an inch from this point, and that a communication existed between its interior and the sinuses of the scrotum. More often inflammation excited in the vermiform appendix by the presence of hard bodies extends to the peritoneum, and either at once induces general peritonitis, or gives rise to adhesions, which, even if gangrene of the part and perforation occur, may prevent the fatal result for some time. Cases have been observed in which a communication has been formed by ulceration between the vermiform appendix and the cæcum, and the foreign body has thus escaped without affecting the peritoneum. We have also seen a case in which a fish-bone, lodged in the vermiform appendix perforated the wall, and also a part of the peritoneum to which it was adherent behind, so as to penetrate into the post-peritoneal tissue, and there produce an abscess, while the peritoneum was uninjured. Pins have sometimes been found in this appendix incrusted with fæcal matter. In one such case the irritation thus caused seemed to have set up a sort of pyæmia and given rise to multiple abscesses in the liver (" Trans. Path. Soc.," vol. xxi. p. 231). Rokitansky mentions a curious accident of a different kind which sometimes befalls the appendix. Its canal gets blocked up at a certain part by a foreign body without ulceration taking place. In consequence of this the mucous secretion accumulates in the closed receptacle, which it distends into a kind of dropsical pouch lined by a thin serous-like membrane.

Affections of the vermiform appendix are very much more frequent in the male sex than the female, in forty-six cases out of fifty-two, according to Dr. Crisp.

Dr. Crisp gives the following enumeration of the foreign substances found in thirty-two cases:—In seventeen, concretions of various kinds; in three, hairs or bristles; in two, bone; in two, a pin; and in the others, a human tooth, a cherry-stone, the shell of a hard nut, fig-seeds and raisin-seeds, while in three it was unknown (" Trans. Path. Soc.," vol. x. p. 152.)

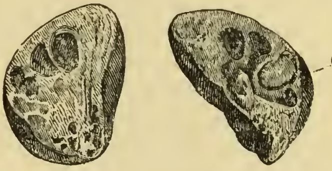
Perforating Ulcer of the Duodenum.—This form of ulcer requires some special notice. It much resembles the round perforating ulcer of the stomach, and being found in a part where gastric fluid must often penetrate probably owes its existence in part to corrosive action. These ulcers are usually circular, sometimes extended in the transverse direction, and rarely surrounding the bowel. They may cicatrize, forming a contracting fibrous scar, which causes stricture of the duodenum or of the ductus choledochus. The edges are often perfectly smooth and without any thickening. They are most frequently situated in the first part of the duodenum, and it has seemed to us, generally on the superior or anterior aspect. Perforation takes place in a considerable number of cases. This may open into the peritoneum direct, producing acute peritonitis, or be partially closed by adhesions of the liver, pancreas or gall bladder; or may, in some cases, penetrate through the soft parts and muscles to the outside of the body, producing a fistula, which is said to open most frequently under the right scapula. Perforation sometimes takes place very suddenly in persons apparently healthy. A case where death occurred in eighteen hours in a presumably healthy and very vigorous man is recorded in the " Pathological Transactions " (vol. xix. p. 236), and a specimen is preserved in the museum of St. Mary's Hospital from the body of a gentleman who was taken ill while walking in the streets, and died almost suddenly from the perforation of such an ulcer. This affection is said to occur more frequently in men than women, in the proportion of five or ten to one.

Ulceration of the Duodenum after Burns.—Mr. Curling, several years ago, observed that in several cases of death from burns, ulcers were found in the duodenum, and other cases have been subsequently recorded. This association of morbid conditions cannot, however, be very frequent, since pathologists with unusual experience, such as Dr. Wilks, have never met with it.

Special Affections of the Rectum.—Lacerations of the rectum and anus occasionally take place, all the coats sometimes being torn through, as after a severe labour when the perineum has quite given way, or only the mucous lining being injured, as sometimes happens after the passage of concretions or hardened fæces. The rectum may be excessively distended by fæcal accumulations, especially in persons of lax fibre and low nervous power, or when paraplegia exists. Sometimes its channel is much narrowed by the pressure of surrounding organs, when displaced or diseased; a retroverted uterus, an enlarged prostate, a vesical calculus, or a pessary in the vagina, may all have this effect. Rokitansky asserts that hypertrophy of the sphincter ani may give rise to obstinate constipation, and even to ileus, and that it frequently induces excoriation of the mucous membrane, the so-called fissure of the rectum. We think the converse is generally, if not always, the case, that excoriation or cracking of the mucous membrane by the irritation which it excites, becomes the cause of excessive action and consequent hypertrophy of the sphincter. This is the opinion also of Sir B. Brodie, who says that "the contraction of the sphincter appears at first merely spasmodic; but in proportion as this muscle is called into action it increases in bulk; and after the affection has continued for some time, it becomes considerably larger." Fissures may be situated at various points as described by Dupuytren; some of which are below the sphincter, and scarcely involve any texture but the skin, occasioning only pruritus. Those which are above the sphincter give to the finger the sensation of a knotty hard cord and during the act of defecation give rise to indescribable tenesmus. They are commonly produced by the ulceration of internal piles, and mark their situation on the cylinder of fæces by a streak of puriform, sometimes bloody mucus. Fissures situated on a level with the sphincter are the worst, being attended with such agonizing pain during defecation, that patients have been known nearly to starve themselves to avoid the recurrence of the action as much as possible. The appearance of these ulcers is that of a narrow fissure, "the bottom of which is red, and the margin somewhat swollen and callous. "Catarrh and blennorrhœa," says Rokitansky, "accompanied by hypertrophy of the coats, which frequently gives rise to plicated and polypous excrescences of the mucous membrane, are very frequent affections of the rectum." Dr. Copland describes *rectal polypi* as varying from the size of a pea to that of an egg, having a broad or very narrow pedicle, situated high up or low down, presenting generally a mucous aspect, a pale reddish hue, and a smooth or lobulated surface. A small growth of this kind, which we had the opportunity of examining had a short pedicle, was of the size of a pea, rather highly vascular, of lobulated aspect. It consisted entirely of Lieberkuehn follicles, and of low folds or ridges covered with well-marked columnar epithelium, and mingled with only a small quantity of fibroid tissue.

Hæmorrhoids depend essentially on a dilated condition of the veins of the rectal mucous membrane, and are in some degree analogous to the common varices of the legs. They are named *internal*, or *external*, according as they are situated above or below the sphincter. Although all take their origin in dilatation of the hæmorrhoidal veins, yet in their subsequent progress they come to present different appearances which we proceed to notice. The first variety sometimes termed *mariscæ*, are described by Dr. Copland "as fleshy tubercles, of a brownish or pale red colour, situate within the anus, or descending from the rectum. They have a somewhat solid or spongy feel ; and when divided they present a compact, or porous, and bloody surface. As the blood oozes from the cut surfaces, they become pale and flaccid." Whether internal or external, they often contain a central cavity filled with fluid, or coagulated blood, of a dark colour. "More frequently there is no regular cavity, the substance of the tumour being as if infiltrated with blood, which becomes coagulated and dark ; but this appearance is not owing to extravasation, but rather to a dilatation of a number of small vessels which traverse the tissue in the direction of the axis of the rectum ; as upon dividing the part longitudinally, numerous dark streaks are seen in its substance, while a section made transversely shows only small roundish specks." These tumours elongate, assuming a conical form with bases larger than their necks. Sometimes blood is exhaled from their surface, sometimes only a serous fluid, and sometimes when they are external they are quite dry. At first they generally disappear in two, three, or four days ; but return again at an uncertain, or at a regular period, and increase in size, becoming firmer in texture. "After some blood is evacuated from them, or after the determination of blood to the parts has ceased, they collapse, leaving small pendulous flaps of skin, which ultimately disappear if the tumours have been small ; but if they have been large, these flaps continue conspicuous, and give a projecting and irregular margin to the anus." Having been strangulated by the sphincter, or repeatedly engorged with blood, or chronically inflamed, these tumours become more permanent and solid. "The permanent state of the tumours is owing partly to the development of capillary vessels, and partly to the effused blood and lymph becoming organized ; this latter circumstance especially giving rise to the excrescences, or irregular mass of tumours found around the anus in those subject to hæmorrhoids." The *second variety* of hæmorrhoidal tumours includes such as are formed by a pure dilatation, or varicose state of the veins of the part. Dr. Copland, from whom we continue to quote, describes them "as not so disposed to enlarge at particular periods, and as more permanent and less painful than" the first variety. "They are commonly of a dark or bluish colour, and soft and elastic to the touch." They are easily emptied by compression, but quickly fill again. "They are round and broad at the base, and often distributed in irregular or ill-defined clusters," which extend often for some way up the rectum, sometimes even as far as the colon. M. Begin observes "that, in most cases, the dilated, superficial, submucous, or subcutaneous veins are only the smaller part of those surrounding the rectum. Sometimes the lower part of this intestine appears as if plunged in the middle of a network of dilated and engorged veins, forming a thick vascular ring, the incision or puncture of which may give rise to dangerous hæmorrhages."

Fig. 138.



A slightly lobulated tumour divided in its middle, and the cut surfaces exposed. It was passed per anum. It seems to have been formed by exudation taking place around varicose dilatations of the veins. The cavities seem to have resulted from the dilatation of mucous follicles.

If in consequence of inflammation or congestion of the varicose vessels, exudations of plastic matter take place around or in the substance of these tumours, they become more solid, and more or less similar to those of the first variety. Sometimes the products of inflammation are deposited within the dilated vessel, which induces its obliteration, and the atrophy of the tumour. Perhaps in some cases the reverse takes place, a vein within an originally solid tumour may become considerably dilated. In short the varieties met with seem chiefly to depend on the predominance of vascular dilatation, or surrounding plastic exudation. A *third* variety of hæmorrhoidal tumours are described as of an erectile character. They are soft and spongy, and of a purplish colour, and give rise to considerable losses of blood. Dr. Colles found in one case "blood-vessels of the size of crow-quills, running for some way down the intestine, then dividing each into numerous ramifications, and each forming by the interlacing of its numerous branches one of these erectile or vascular tumours. The trunks and branches of these vessels were covered only by the lining membrane of the intestine." Some hæmorrhoidal tumours appear to result from the effusion and coagulation of blood in the surrounding tissue. The reality of this occurrence is denied by Rokitansky, but we quite acquiesce in the remarks of Mr. H. Lee,* that it is by no means easy to determine whether the delicate, smooth, and shining membrane lining the cavities, in which the coagula are contained, is the lining membrane of the venous system, or one of new formation derived from the blood itself.

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* "Med. Gaz." Aug. 1848.

He also notices the effect of hæmorrhoidal tumours on the mucous membrane around them, which is raised and forced down along with them when they are protruded beyond the sphincter, so that at last it becomes permanently relaxed and "baggy." The female sex, sedentary occupations, and constipation, are enumerated as the chief cause of hæmorrhoids, to which we should add a plethoric habit, and a lax condition of fibre. It does not appear at all proved that cirrhosis of the liver, or obstructive disease of the heart, has any marked influence in the production of piles, as one would naturally expect. The mucous membrane thinned over an hæmorrhoidal tumour is prone to ulcerate, and the resulting sore, according to Rokitansky, is characterized by its seat in the vicinity of the sphincters, its irregular shape, its indented and sinuous flabby margin of mucous membrane, and the ridges of similar tissue that surround or pass over it. These ulcers may continue to burrow into the surrounding areolar tissue, and give rise there to abscess, and ultimately to fistula in ano.

Fistula in ano.—This, however, more often results from inflammation being set up in the deeper-seated tissues, the areolar and adipose, but still in the vicinity of the bowel, which advances to suppuration, and in most cases makes its way first outwardly through the integument surrounding the anus, and afterwards establishes a communication with the cavity of the rectum by a small aperture situated very constantly at the distance of an inch, or an inch and a quarter, from the anus. It does not clearly appear why fistula in ano should occur so often as it does in persons prone to pulmonary tuberculosis, and still less why its existence should be preservative, at least in not a few cases, against the invasion of the dread malady. *Cancerous disease* attacks the rectum in most of the forms mentioned as affecting the whole intestine; the only one which it seems desirable especially to notice, is that which gives rise to the annular stricture. This occurs almost exclusively at the upper portion of the rectum, especially at the junction of the sigmoid flexure; the strictured part is sometimes unattached, more often firmly agglutinated to the promontory of the sacrum, but is nevertheless pushed down by the fæcal accumulations above, so as to be within reach of examination by the finger.

ABNORMAL CONDITIONS OF THE INTESTINAL CONTENTS.

Intestinal Gases.—The accumulation of an excessive quantity of *gas* in the intestinal canal constitutes the most ordinary form of Tympanitis. It often occurs in inflammatory affections of the canal, which induce more or less paralysis of the coats, in consequence of which the gas is not expelled, but goes on accumulating. The gas in the stomach of an executed criminal was found by Magendie and Chevreul to consist of atmospheric air with a part of its oxygen replaced by carbonic acid, and some hydrogen. In cancerous strictures of the pylorus, and in chronic catarrhal states, the gas contains but little oxygen, much carbonic acid (probably also hydrogen and carburetted hydrogen), and constantly also sulphuretted hydrogen. In the small intestines of criminals Magendie and Chevreul found an abundance of hydrogen and carbonic acid, no oxygen, and a varying quantity of nitrogen. Marchand found in the gaseous contents of the large intestines carbonic acid, nitrogen, hydrogen, carburetted hydrogen, and a small proportion in one case of sulphuretted hydrogen. That these gases are secreted by the mucous lining, and do not proceed from decomposition of the ingesta, is considered improbable by Lehmann. We cannot, however, coincide in his opinion, at least to the exclusion of the first-mentioned way of production, if for no other reasons, on account of the experiment performed by Frerichs, which he himself details, that a portion of intestine emptied of its contents, and isolated from the rest of the canal by two ligatures, always became full of gas after being left some time. Clinical experience also points strongly to the same conclusion. The other view, however, is maintained by all recent investigators.

According to the accurate and classical researches of Planer, the gases of the stomach appear to be chiefly air swallowed with the food, which has lost oxygen and gained carbonic acid, possibly mixed with a small proportion of gases of decomposition. Thus they are chiefly nitrogen and carbonic acid with a small proportion of oxygen.

In the small intestine the oxygen fails entirely, but a small proportion of hydrogen appears, derived from the decomposition of food. In the large intestine are found nitrogen, carbonic acid, some hydrogen, and hydrocarbons, chiefly marsh gas, and traces of sulphuretted hydrogen, but the proportions of these depend very greatly upon the food.

	Stomach. (Planer.)	Ileum. (Planer; post-mortem.)	Colon (after Ruge) obtained during life.
N	68·68	79·73	17·50
CO_2	25·20	16·23	40·51
CH_4	—	—	19·77
H	—	4·04	22·22
H_2S	—	—	traces.
O	6·12	—	—

Stagnant gas simply contained in any cavity of the body soon comes to consist almost entirely of nitrogen and carbonic acid.

Planer found fifty per cent. of nitrogen in the gas accumulated in the colon above a structure in the sigmoid flexure.

Concretions are occasionally met with in the intestinal canal, and in rare instances of very large size. Dr. Monro (Primus) observed some varying from five to eight inches in circumference, and Monro (Secundus) removed one from the colon which weighed four pounds. Sometimes several, as many as thirty, exist together, but commonly there are not more than two. The colour of the smaller resembles that of iron ochre, the larger are more of a coffee colour, and occasionally whitish. "They are generally found in concentric layers, and are often radiated, sometimes very obscurely, from nuclei. They are more or less porous, either spheroidal or oblong, and vary from the size of a pea to that of a hen's egg, or still larger." The nuclei of concretions may be gall-stones, fragments of bones, fruit, seeds, &c., round which saline and animal or undigested matters collect and become condensed. In Scotland where oatmeal is much used as an article of food the fibres of the husk of the oats have been found to constitute a large part, or nearly the whole of the concretion; and in a similar manner chewed paper, the several portions being matted together by mucus with fæcal and earthy matter, has been known to cause their formation. Such concretions often exhibit no distinct nucleus. Concretions, which may be mistaken for gall-stones, but which are of a fatty nature, are sometimes voided by persons who suffer from a torpid state of the bowels, and deficient digestive function. Dr. Copland describes them as of a globular form, varying from the size of a pea to that of a large grape, of a cream colour, slightly translucent, and of the consistence of soft wax. It is probable that these concretions, as well as the evacuations of a more fluid fat which occasionally take place, are connected in some measure with disease of the pancreas. Observation of disease has in several instances shown the coincidence of the two phenomena, and Bernard has offered a physiological explanation, viz. that the use of the pancreatic secretion is to make the fat contained in the food capable of being absorbed. His results, however, are denied by Frerichs, Bidder, and Schmidt. *Blood* is sometimes effused in greater or smaller quantities into the intestinal canal. This may result from active or passive hyperæmia, ulcerations, purpura, scorbutic dysentery, and we have once seen it in a case of death from a severe fall.

The parasites of the intestines have already been enumerated.

Mucus in any appreciable quantity can scarcely be said to exist in perfectly healthy intestines; but it is secreted abundantly, as we have seen, under catarrhal irritation. Rokitansky applies to it the following epithets in various cases; it is either milky white, yellowish, and purulent, or glutinous, transparent, vitreous, spawny. He also states that there can be no doubt that a peculiar gelatinous constitution of the mucus is the nidus of intestinal entozoa, and favours helminthiasis. Under irritation of an acute character small membranous patches of mucus are often passed; we have seen these in a case of dysentery constituting the whole of the scanty evacuation. Between these and the fibrinous tubular formation before noticed there is no very essential difference, and both are to be distinguished from certain membranous substances which occasionally appear in the evacuations, and may cause some perplexity to the patient and physician, unless their nature be understood. These are simply the undigested remains of some tendinous expansions which contain a great deal of yellow elastic fibre, on which the gastric juice seems to act with much less energy than on the white.

CHAPTER XXXVI.

ABNORMAL CONDITIONS OF THE LIVER.

CONGENITAL malformations are rare; absence of the liver is only observed in extreme cases. Sometimes the left lobe retains, in a greater or less degree, its fœtal proportion to the right.

Umbilical Vein.—This vessel, so important in fœtal life, which becoming obliterated forms the round ligament of the liver, sometimes remains pervious, though very small, and forms a minute channel leading from the portal vein to a superficial vein or sinus in the abdominal wall near the umbilicus. Often a channel may be traced in the ligament from the portal vein for about three or four inches, then becoming obliterated. These conditions may become important when in diseases of the liver, as cirrhosis, the portal circulation is obstructed, since communication may thus be opened up between the portal and systemic circulations. It has been generally assumed that this communication must be through a reopened umbilical vein, but Sappey showed that the appearance of such a vein might be produced by the enlargement of certain small accessory veins, shown by him to run within the suspensory ligament of the liver, forming an anastomotic network round the obliterated umbilical vein, and opening either into the still pervious portion of this, or into the portal vein. In several cases of cirrhosis observed by the editor, the explanation of Sappey has proved to be correct; but in one case a vein representing the umbilical vein was found to be continued as far as the umbilicus, with a calibre equal to that of a swan-quill.*

Acquired Malformations.—The practice of tight lacing in women is sometimes the cause of marked deformity in the organ. The lower ribs of the right side are pressed inwards so as to indent the outer aspect of the right lobe, causing great convexity, while the lower margin of the lobe projects below them. Sometimes the lower margins of both lobes are simply compressed and approximated, so that the whole organ becomes bluntly conical or almost heart-shaped. These changes are now scarcely seen, except in the bodies of old women.

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* Henle, "Handbuch der Anatomie," vol. iii. pp. 371, 388. Sappey, "Mémoires de l'Acad. de Médicine," vol. xxiii. p. 269. Champneys, "Journal of Anatomy and Physiology," vol. vi. p. 417.

In men the practice of wearing a tight waist-belt may produce a broad white band, with thickening of the capsule, passing horizontally across both lobes.

CONGESTION.

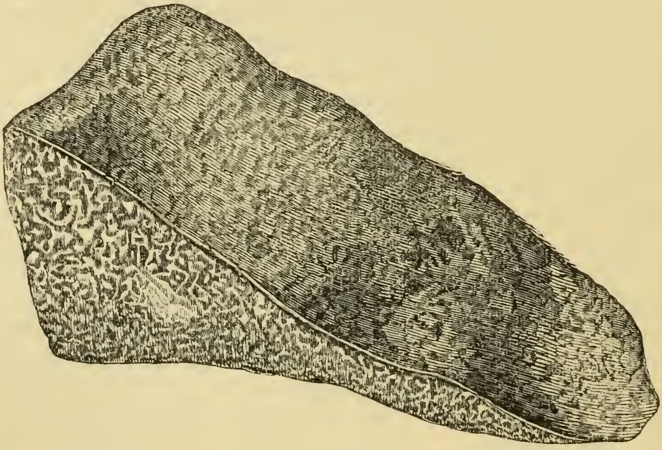
The vascular apparatus of the liver is very large, its capillaries are more capacious than those of most other parts, and the calibre of the portal and especially of the hepatic veins is extremely ample. It is, therefore, capable of containing a very large quantity of blood. Though its surface is closely invested by its capsule, yet this membrane allows of a good deal of distension, and this is also attested by the tortuosities of the arterioles of the surface, which probably exist for the purpose of allowing the vessels to be elongated without injury to their texture. Congestion of the liver may be general or partial. The latter is far the more common condition, and, indeed, in its lower degrees is not morbid. The central parts of the lobules, in perfectly healthy livers, are often seen to be marked by a spot of redness which occupies about the middle two-fourths of the whole diameter. This is produced by the blood, as the circulation gradually failed, having stagnated in the hepatic veins, in their radicles, the intra-lobular veins, and the surrounding capillaries also. Mr. Kiernan named this, "Hepatic Venous Congestion of the first degree." When the congestion extends further in the direction backwards, there are seen no longer mere spots of redness, but patches of very irregular shape surrounding, more or less completely, portions that are not congested. These portions are situated at the interlobular spaces, where three or more lobules adjoin. Such a condition was named by Mr. Kiernan, "Hepatic Venous Congestion of the second degree." This may coexist with a perfectly healthy condition of the cells which occupy the meshes of the capillary plexus, but not unfrequently these are variously altered, and thus give rise to some modification of the appearance. The appearance of the liver greatly depends upon contrast between the central and peripheral portions of the lobules. This is most marked in the so-called "*nutmeg*" condition.

Consequences of Congestion.—Congestion of the liver, although extreme, does not seem to occasion any structural change, if it is only temporary; but, if, as in the case of obstructive cardiac disease, it results from a permanent cause, and is, consequently, itself permanent, it produces the following effects. The distended capillaries, especially the hepatic portion, press on the intervening cells; these become, in part, atrophied or stunted—in extreme cases, almost destroyed: in part, they are gorged with yellow matter to such a degree that they appear as opaque masses. The quantity of yellow matter thus formed is far greater than any that exists in healthy states of the organ, and, as some of it is doubtless absorbed and carried into the blood, we find in this circumstance some explanation of the icteric hue which is so often observed in such patients. The connection which certainly exists between the congestion and the yellow engorgement of the cells, as cause and effect, gives additional support to the opinion that the yellow bile-pigment is a modification of, and derived from, the colouring matter of the blood.

Like venous congestion in general, congestion of the liver from cardiac disease, produces first enlargement, which may often be recognized during life, and later on wasting, dependent, doubtless, on the imperfect nutrition of the organ. With this is associated some amount of induration, though less than is produced by some other causes. Generally at the *post-mortem* examination we find the liver rather small than large, hard, and with the characteristic nutmeg appearance.

Chronic Congestion, Nutmeg Liver.—This consists, in its best marked instances, of deep reddish-brown congestion, forming patches and streaks occupying the central parts of the lobules, and surrounded by patches of a greyish, or yellowish white colour. The congested portions are most definitely limited, and the contrast between them and the pale parts is extremely striking. This partly depends on the circumstance that the portions thus devoid of blood are affected with fatty degeneration or infiltration, a change which, by causing the cells to increase in bulk, occasions

Fig. 139.

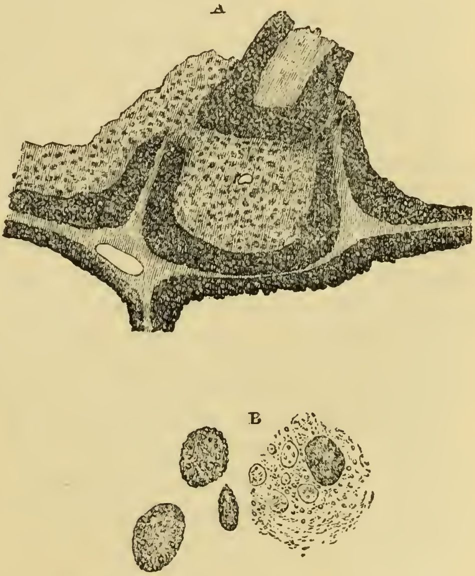


Section of liver showing the nutmeg appearance; the dark parts are the deep red congested central parts of the lobules.

compression of the intervening capillaries. That this is the true cause of the limitation of the congestion is proved by its exactly ceasing at the inner margin of the zone of fatty degeneration. Generally speaking, fatty change begins in the peripheral parts of the lobules, and it would seem that these portions contain physiologically more fat than the central portions. Many of the cells in the congested part are seen filled with dark yellow matter; very many, also, are atrophied, probably in consequence of the blood stagnating in the distended capillaries, while some granular pigment is diffused in the surrounding parts. The nutmeg appearance may be exhibited in some degree by livers which are quite free from fatty degeneration, but it is never so marked as in the condition just described.

The correctness of this explanation may be shown by cutting a thin slice of the liver and putting it under a low power of the microscope, or simply holding it up to the light, when the dark central portions will be seen to be comparatively transparent, while the peripheral portions are opaque from the abundance of fatty cells.

Fig. 140.



(A) Section showing lobules of the liver, bounded by marginal zones in a state of fatty degeneration. The interior of the lobules is deeply congested, and contains much dark yellow pigment in masses, but is less opaque than the marginal portion.

(B) Cells loaded with pigment, atrophied cells and granular matter from the interior of the lobules.

When the venous congestion becomes more general, occupying every part of the liver, the organ presents a deep red colour throughout, though, even in this case, the centres of the lobules present the darkest tint; it is also enlarged often to a greatly increased size, and becomes more firm and prominent, so as to be readily perceptible to the touch, below the margin of the ribs, on the right side. The result of injecting a liver which is nearly drained of its blood, as one taken from a slaughtered sheep, is very instructive as to the amount of enlargement that may be produced by congestion. The organ swells up as the fluid is thrown in, and when fully injected is of nearly double its former size, greatly more dense and solid, with its thin anterior margin prominent and hard. Sappey found on weighing livers with the vessels empty, and afterwards filled with water by injection, that there was a gain of weight amounting on the average to one-third. Such a state may well produce a sense of weight and fulness in the right hypochondrium.

Causes of Congestion.—The most frequent cause of congestion to any degree that can be considered morbid is organic disease of the heart, especially such as produces great obstruction to the circulation, and throws the blood back upon the right side of the heart, and the venous system in general. All causes of apnœa produce the same effect, and act in the same way. Congestion thus induced is passive; active congestion takes place in inflammation of the liver, or, as we have observed, when large doses of calomel are administered. Active congestion may be produced also by various irritants, as alcohol, and is in some degree physiological, as an accompaniment of the digestive process. The congestion which occurs in the cold stage of ague and other fevers seems to depend not solely on the recession of the blood from the surface, but chiefly on paresis of the vasal nerves of the organ.

Portal venous congestion is a rare variety of partial: the centres of the lobules are pale, and are surrounded by continuous red zones. It is said to occur in children only.

Hœmorrhagic effusion may take place as the result of extreme congestion, but this is rare; the blood may be poured out either on the surface, detaching the capsule for some extent, or deeper in the substance of the organ. The former occurrence Rokitansky states is most common in new-born infants, and may even proceed to such an extent as to rupture the serous investment, and allow the escape of blood into the peritoneal cavity. The deep-seated extravasations occur more frequently in adults than the superficial, and constitute apoplectic spots of various forms and size. After their partial absorption a fibrous cicatrix remains.

Hæmorrhage in the substance is sometimes seen in scurvy or purpura hæmorrhagica. Severe bleeding into the peritoneum follows rupture of the liver, and may be the immediate cause of death. Nevertheless, even this may be healed, and recovery take place.

INFLAMMATION OF THE LIVER.

Inflammation may affect either the substance of the organ, chiefly *Hepatitis*, or its capsule *Perihepatitis*. The former is, in temperate climates, a rather rare affection, the latter extremely common. The only well-marked form of acute inflammation of the liver is the purulent. Other affections, sometimes called inflammation, appear to be better described as degenerations. Purulent hepatitis, or abscess, occurs in two forms—(1) solitary, or tropical abscess; (2) multiple, or pyæmic abscesses.

1. *Solitary, or Tropical Abscess.*—The former rarely occurs in this

country, except in persons coming from tropical climates. It is common in India. The early stages of this affection are not well known. When suppuration has occurred an abscess will be found, varying in size, sometimes involving nearly the whole of the liver; its inner surface either ragged or lined by a smooth "pyogenic" membrane. The surrounding tissue may show no change except simple congestion. This form of abscess occurs more often in the right lobe than the left. It may open into the peritoneum or pleura; or more rarely into the stomach or intestine. By opening into the bile ducts the contents sometimes become tinged with bile.

The solitary abscess of the liver is in the majority of cases connected with ulceration of the bowels; usually with dysenteric ulceration of the colon; and hence its infrequency has some relation to the rarity of the severe form of tropical dysentery in this country. The abscess is generally regarded as the consequence of the ulceration; though some authorities, as Dr. Murchison, take the converse view, believing ulceration to be not a constant accompaniment of hepatic abscess, and when it occurs, secondary to the latter. There are, however, many cases on record of hepatic abscess secondary to various kinds of ulcers in the intestines; which seem to connect the form we are now considering with pyæmic abscesses strictly so called.

Solitary abscess sometimes occurs independently of dysentery or ulceration; and has been referred to cold, excessive drinking of spirits, and other causes.* A similar abscess may result simply from direct injury, such as, according to Dr. Wilks, may be inflicted by a fractured rib. Some cases are quite unexplained. Finally, a single large abscess may be formed by the confluence of several smaller, or of multiple pyæmic abscesses, so that the distinction between the single and multiple abscesses may disappear; and some authors, as Dr. Moxon, contend that both classes originate in the same way, the large, or tropical abscesses, differing only in being more chronic. But the descriptions given of the tropical abscess represent a disease very different from pyæmia.

2. *Multiple Abscesses of the Liver.*—These are chiefly the result of pyæmia; other occasional causes will be pointed out hereafter. The pyæmia may be either limited to the parts connected with the portal vein, or general. In the former case some morbid material appears to be absorbed by one of the veins tributary to the portal trunk, connected with some diseased part, thus carried into the branches of this vein within the liver. The transport is in many cases effected by masses derived from softening thrombi, but very often nothing of this kind can be discovered. In cases of general pyæmia the morbid material must be conveyed through the arteries till it becomes arrested in the portal capillaries. In either case the result is not merely mechanical, due to the obstruction of capillaries, but specific, due to the inflaming properties of the morbid matter.

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* Murchison, "Trans. Path. Soc.," vol. viii. p. 237; Bristowe, Ibid., vol. ix. pp. 241, 273.

It was formerly supposed that abscesses in the liver were specially likely to be the consequence of injuries to the head; but this appears to have been merely an error of observation, caused by not taking into account a sufficient number of cases. Injuries to the head are more liable to produce pyæmia than injuries to some other parts; but the pyæmia thus produced has no special tendency to attack the liver: abscesses in the lungs being, as in all other cases, of much more frequent occurrence.

Multiple abscesses may also result from suppurative inflammation of the bile ducts; which sometimes, though not always, extends into the surrounding tissue, producing abscess. They may also be the consequence of changes occurring in softened thrombi contained in the larger portal-vein branches which are spoken of below.

In a few rare cases diffuse suppuration of the liver, producing numerous small abscesses, but not apparently due to pyæmia, has been observed. One of the best instances is described and figured by Dr. Quain, in the "Pathological Transactions," vol. iv. p. 171, plate v.

Pyæmic abscesses are sometimes preceded by a short stage of diffuse suppuration, when the form of the lobules can still be recognized, though their substance is very soft, and of a yellowish colour. The commencing abscesses are at first very small, like spots of purulent matter dispersed here and there through the inflamed and softened tissue; they gradually enlarge, several coalesce together, and thus form cavities of irregular shape and size. The parietes of the abscess are uneven, presenting the remains of former partitions, they are covered by a kind of pyogenic membrane which consists, in great part, of pus globules; external to this, the wall is formed by hepatic tissue, infiltrated with exudation matter, which serves to bound and limit the abscess, as in the case of a common phlegmon. In very small abscesses, and those of very recent formation, as also in cases where the process is very rapid, this limitation of the abscess by exudation substance does not take place; on the other hand, in old abscesses, and those of very large size, occurring in tolerably healthy systems, a strong enveloping cyst may be found, consisting of fibroid tissue, and amounting sometimes to three or four lines in thickness.

Consequences of Hepatic Abscess.—If the abscess of either kind be deep-seated and encysted, it may continue, especially if of small size, for a long time without increasing much, or producing serious disturbance of the health, but if it be near the surface it excites inflammation of the serous investment, and effusion of lymph, which soon unites its wall with the part with which it is in contact. This may be the wall of the abdomen, or some of its contained hollow viscera, or the diaphragm which roofs it in above, and by any of these various routes the abscess may extend, and at length discharge its contents. By extension, also, in the parenchyma of the liver, the abscess may reach a branch of the portal vein or of the hepatic, excite inflammation of its coats, and consequent obstruction of its canal by coagulated fibrine. When the enlarging abscess reaches an hepatic duct-branch it affects it in a different way—it does not set up inflammation in its walls and cause its obstruction (though this may no doubt occur in some of the smaller); but it ulcerates through its tunic, and establishes a communication between the efferent channel and its own cavity. Hence it occurs that the pus contained in large abscesses is always mingled with a considerable quantity of bile, while that of the smaller and recent abscess is almost pure. An abscess of the liver may sometimes heal either after the evacuation of its contents, or after they have been absorbed; the latter occurrence is, we should conceive, exceedingly rare. In either case the walls of the abscess approach each other, and at last collapse together, including sometimes a quantity of solidified purulent matter, which at a later period forms a cheesy or cretaceous mass, attesting by its presence in the midst of the cicatrix the nature of the changes which had previously taken place.

Distinction of Pyæmic Abscesses.—When several abscesses are present, there will be little difficulty in coming to a conclusion; when one only, it will if pyæmic be usually recognized by the zone of congested tissue surrounding it, by occurring in the midst of a block, partially softened, by its extremely ragged, irregular outline, and the absence of a distinct wall; unless, indeed, it have existed for some time, in which case more than one will probably be present. Also of course the changes in other organs and the history will assist the distinction. Hydatid cysts sometimes suppurate, but their other characters usually distinguish them sufficiently.

Perihepatitis.—The capsule of the liver is very often indeed attacked with inflammation, or presents, on post-mortem examination, such changes as are usually ascribed to this process. Bands of adhesion of various length, and attaching different parts of its surface to contiguous organs, are very often found: these are pretty certain evidences of an acute or subacute by-past hepatitis; they are often traversed by newly-formed vessels which establish a communication between the capsular arterioles and the contiguous vessels of the general system. In a specimen which we injected, the vessels of a patch of false membrane, on the surface of the capsule, were seen to be of much greater calibre than the vessels in the adjacent healthy part. Adhesions commonly form, as said, over superficial abscesses of the liver: it is rare that this fails to take place, and that an abscess bursts into the peritoneum. Over hydatid tumours and cancerous masses they are less frequently formed. Dr. Budd particularly notices their absence, and considers that their production is the exception and not the rule. This may, perhaps, be in some measure accounted for by another circumstance relative to cancerous tumours in this organ mentioned by the same observer, viz., that they seem capable of tainting with their own peculiar morbid action opposed parts with which they come in contact; such tainting of a part may be conceived to be quite inconsistent with the effusion of comparatively healthy lymph. The capsule of the liver is very frequently found thickened, appearing whiter, more opaque, and dense; this thickening generally occurs in patches of various size, which sometimes coexist together with the bands of adhesion just noticed, but quite as often are independent of them, or coincident with similar thickenings of other serous membranes. It seems doubtful whether these changes should not be more properly classed as degenerations than as inflammations.

One conspicuous form of perihepatitis is produced by syphilis. The capsule is found greatly thickened, often over a large extent, and the liver wasted. A considerable degree of perihepatitis also often accompanies cirrhosis. Affections of the capsule of the liver are, however, usually associated with similar affections of the peritoneum generally. Small fibrous outgrowths, called by Dr. Moxon "Pacchionian bodies of the Liver," are sometimes found in association, it is said, with obstructive disease of the heart, especially of the right side.

Gangrene of the liver is very rare; Rokitansky well observes that "it is developed in parts affected with inflammation and suppuration, not so much as a result of intense inflammation as of certain peculiar conditions which cause a tendency to gangrenous degeneration." These peculiar conditions may be probably either low, unhealthy states of the general system, or, as in the interesting case recorded by Dr. Budd, the septic influence of a previously healthy part which had been affected by gangrene.

Cirrhosis, or Chronic Interstitial Hepatitis.*—This condition, which is sometimes described as chronic inflammation, consists essentially in an hypertrophy of the connective tissue framework of the organ, with degeneration of the parenchymatous substance. It occurs in two forms, very different in external appearance, though essentially the same—(1) The granular or contracted form, *cirrhosis* as usually understood; (2) The smooth form, sometimes recognizable with difficulty except by microscopical examination.

1. *Ordinary Cirrhosis.*—A liver which is affected by this change in an extreme degree is remarkably altered. It is much smaller than natural, much paler, and instead of presenting a smooth surface, is contracted and puckered so as to resemble, according to a former observer, "a congeries of little firm globules like the vitellarium of a laying hen." These globular portions are yellowish or bile-stained, and project on the surface; the same are seen on section.

They are of various sizes, and evidently consist of parenchymatous substance: they are surrounded, and as it were capsulated by firm fibroid tissue which extends throughout the whole liver, and gives it a remarkable degree of density and firmness. This fibroid tissue is at first highly vascular: it is evidently a new formation, and as such tissue frequently does, contracts and shrinks together, and so draws in the surface at various parts as to produce the irregular nodulated, or "hob-nail" condition, as it is familiarly termed. The same shrinking also affects the vessels which supply the liver: they are surrounded and ensheathed by fibrous tissue in the healthy state, and when this is morbidly thickened and condensed, the pressure exerted upon them narrows their channels and materially diminishes the quantity of blood which they are able to convey. Hence the portal current is checked at its very origin, and the congested capillaries are obliged to relieve themselves by effusion of serum into the peritoneal cavity. The capsule of a cirrhotic liver is sometimes smooth, sometimes thickened or attached by adhesions to adjacent parts: these adhesions are often traversed by newly-formed vessels, which form a kind of collateral circulation between the portal vein and the general system.

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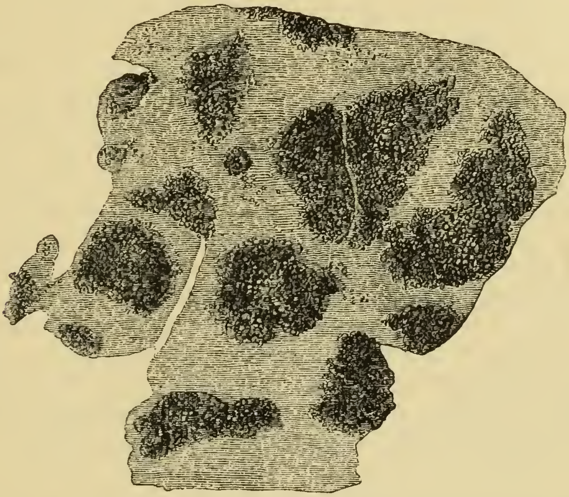
* Cirrhosis is derived from κιρρός = orange-coloured or tawny, between red and yellow (Liddell and Scott);—from the colour of the degenerated parenchymatous portions.

Fig. 141.



Section of liver in a fatty state, with abundant new formed fibrous tissue between the lobules.

2. *Smooth Interstitial Hepatitis.*—Such is a brief description of a liver affected with ordinary cirrhosis; but there are other conditions of the organ, essentially similar, in which the external appearances are very different. The organ is firm and dense, evidently from the presence of an increased quantity of fibroid tissue, but its surface is not puckered or but slightly, nor its edges

rounded; it may be pale or else highly congested with blood, usually of the "hepatic venous second degree," and is often very considerably enlarged. It is sometimes of a greenish colour from bile-staining. Microscopic examination shows that the quantity of fibroid tissue forming the Glissonian sheaths is considerably increased, sometimes to such a degree as to encroach on the lobules to a great extent, and produce atrophy of their substance; it is at the same time thickly infiltrated with small cells or nuclei. It is possible that some livers which present these appearances may be in an early stage of cirrhosis, and would subsequently become contracted and nodulated; but we strongly incline to the belief that this is not the case with the majority, and that the morbid alteration is somewhat different. The difference probably consists in the more general and extensive formation of fibroid tissue throughout the liver, in its being less confined to the portal canals. It is rather remarkable that the hepatic cells in extremely contracted cirrhotic livers sometimes present a tolerably healthy aspect; their nuclei are distinct, and though, perhaps, containing less oil than usual, they are by no means destroyed or seriously altered. They are far more affected in the dense, firm, uncontracted livers, partly as the result of congestion with impletion of yellow matter, partly by atrophy from the encroaching fibroid tissue, and these often contain many fatty molecules. The minute ducts which run in the smaller portal canals and between the lobules are often atrophied by the pressure of the condensed fibrous tissue, so that they can no longer be detected; in this way the biliary secretion may be materially interfered with.

It seems to be proved by the observations of Dr. Bright and Dr. Budd that in the early stage of cirrhosis the liver is enlarged; this would seem to depend on the effusion of serum and production of lymphatic corpuscles within its texture during the existence of inflammatory action, as well as on positive increase of the fibrous element. In many cases there can be little doubt that this is the case, and that the cirrhotic change results from a subacute inflammatory action being set up in the Glissonian sheaths. But in many cases we are disposed to think the process is different, that both in the finally contracted and uncontracted livers the fibrous tissue is hypertrophied and condensed rather by a degenerative action than by one which can be termed inflammatory. The change seems to be of a similar kind to that which produces cartilaginoid induration of the capsule of the spleen, stiffening of the valves of the heart, and contraction of its orifices, which can scarcely be regarded as of inflammatory origin. We are confirmed in this view by having often observed various minor degrees of condensation and thickening of the Glissonian sheaths in cases where there was no trace of inflammatory action, though there was decided atrophy.

Causes of Cirrhosis.—The remote cause of cirrhosis, in many cases, is certainly the habit of spirit-drinking: the alcohol absorbed into the portal blood first passes through the liver, and very probably exerts some action on its tissue. This suggests the idea of the spirit acting as a local irritant, and with this Dr. Percy's observation of the greater affinity of alcohol for the liver than for other organs in animals poisoned by it seems to harmonize. Obstructive cardiac disease is probably not a direct cause of cirrhosis in any of its forms or degrees, but certainly must be a predisposing one. Congestion favours the occurrence both of inflammation and of degeneration. Both the heart disease (*e.g.* constriction of the mitral orifice), and the cirrhotic change in the liver, are sometimes, perhaps, common results of the same condition of the blood, or of some general constitutional tendency. There are, no doubt, other exciting causes besides the ingestion of alcohol, but they can scarcely be particularized. We have seen typical cirrhosis in a child three years old, where spirit drinking was out of the question. One form of syphilitic disease of the liver much resembles cirrhosis; but some cases are, no doubt, unexplained.

Consequences of Cirrhosis.—The obstruction to the blood current in the portal vein causes congestion of the whole portal system with the following consequences:—(1) Swelling of the spleen (not invariably); (2) Effusion of serum into the peritoneal cavity, or ascites; (3) Catarrh of the stomach, and sometimes hæmorrhage; (4) Congestion of the mucous surface of the intestines, sometimes producing diarrhœa; (5) Enlargement of collateral venous channels. Ultimately secondary congestion of the systemic venous system may ensue, producing anasarca of the legs and general dropsy.

The spleen, albeit exposed to the backward pressure of the blood retarded in the splenic vein, does not always become distended in the way that one would expect, but is often, on the contrary, sometimes small and soft. Frerichs found enlargement of the spleen in half his cases. We have found it enlarged in nearly all. Ascites is, perhaps, the most constant phenomenon, but may be wanting if the collateral venous channels are abundant. Jaundice, it should be remembered, rarely occurs, and the urine, though always high-coloured, does not contain bile pigment.

Collateral Circulation.—All or most of these consequences may, at least for a time, be averted, if the collateral venous channels be greatly developed. There are, beside the umbilical vein and its attendant veins already mentioned, the following plexuses:—(1) The hæmorrhoidal plexus, forming a communication between the inferior mesenteric and internal iliac veins, and other plexuses in the pelvis which have a similar double connection. (2) Several groups of minute veins running in the ligaments of the liver, distinguished by Sappey in five groups: (*a*) in the gastro-hepatic ligament; (*b*) surrounding the gall-bladder and entering the liver on each side; (*c*) running in the walls of the portal vein, hepatic artery, and bile duct; (*d*) running in the suspensory ligament from the middle of the diaphragm to the liver; (*e*) veins descending from the abdominal wall above the umbilicus by the suspensory ligament to the left longitudinal fissure of the liver. At their upper origin they stand in communication, on the one hand, with the mammary and epigastric veins; on the other, with the superficial veins of the abdomen. Some of this group surround the obliterated umbilical vein, and sometimes communicate with it, as before mentioned.

In a case in which, with well-marked cirrhosis, there was an entire absence of ascites, we observed plexuses connecting the capsule of the liver with the supra-renal veins, and thus with the veins of the diaphragm; on the left side also with the renal vein. As several of these collateral channels open into the azygos veins, these vessels are always found enlarged, of course, especially the right, and convey a considerable proportion of the accessory circulation.

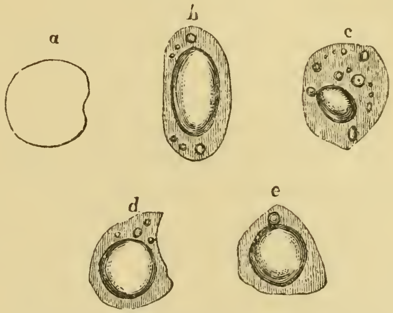
DEGENERATIONS OF THE LIVER.

Under this head we include—(1) Fatty degeneration; (2) Parenchymatous or albuminous degeneration; (3) Lardaceous degeneration; (4) Pigmentary degeneration.

(1.) *Fatty Degeneration.*—The first degenerative condition of the liver which we shall describe is that of fatty degeneration. A liver thus affected is usually much enlarged, paler than natural, and in most cases softer; sometimes, however, it has a feeling of great solidity. The capsule, in cases of uncomplicated fatty degeneration, is not thickened, nor attached by adhesions to adjacent parts. The thin edge of the organ is somewhat rounded, and the thickness generally increased. On microscopic examination, it is at once seen that the hepatic cells are engorged with oil; instead of containing a few minute drops imbedded in a mass of granulous matter, they are filled to the extent of one-half or two-thirds, or even their whole cavity, with colourless fluid oil. Sometimes a quantity of yellow matter is also seen in the cell-cavity, together with the oil, but this is often absent. The nucleus disappears, as is generally the case in cells that have fulfilled their work of secretion, but the envelope persists, and is sometimes a little thickened and striated. In very advanced cases the cells are not found merely gorged with oil, but, to a great extent, broken up and lost; in their place there are seen only granular *débris*, entangling multitudes of oil drops of different sizes. There seems to be less tendency to the development and growth of young cells than in the healthy state, those that are forming appear stunted, and many become very early affected with oily deposit. This seems to show that one very important function of the liver in such cases is, at least, very imperfectly discharged. It is an interesting fact, that the bile in these cases undergoes no constant or necessary alteration; it is sometimes unusually pale, but often has the dark green tint of ordinary bile. The minute ducts are tolerably natural, and doubtless continue to discharge their part of the bile-secreting process. A fatty condition may coexist with a considerable degree of cirrhosis. The appearance of a section under the microscope is then very remarkable; the lobules appear as opaque islets separated more or less widely from each other, by more transparent spaces of fibroid tissue.

The fatty liver is usually much enlarged; in fact, no cause produces such enlargement except cancer. The fatty change is usually confined to the margins of the lobules, and is always, we think, most advanced there; sometimes, however, it may commence in the centres of the lobules. This somewhat rare condition of central fatty engorgement has been explained by rapid removal of fat from the outer portions of the lobules. The pale condition of the liver depends on the enlarged size of the cells, which are pressed closer together, and thus constrict the capacious capillaries and allow less blood to be contained within them. There is, however, no obstruction to the flow of blood, such as we have seen in cirrhosis; the soft state of the oil-laden parenchyma sufficiently accounts for this. We think it most probable that fatty degeneration does not consist merely in the impletion of the cells with oil, in their containing a greater quantity of this matter than they naturally would, but that there is an actual impairment of their development and nutrition, of their active power of formation and secretion, so that the tissue really undergoes a kind of decay. In support of this view, we may mention that we have scarcely ever found sugar present in the parenchyma of a thoroughly fatty liver, while it can almost always be detected in livers that are not so affected; this is certainly a remarkable fact.*

FIG. 142.



(*a*) Empty envelope of an hepatic cell from which the oil has escaped.

(*b*) (*c*) (*d*) (*e*) Hepatic cells containing much oil.

Causes of Fatty Degeneration.—The proximate cause of the production of fatty liver is, we believe, the existence of an undue quantity of oily matter in the blood, in proportion to the assimilative power. In cases of disease it is also very probable that the vital power of the hepatic cells is much lowered. It may, therefore, occur: (1.) When an animal is largely fed on food containing much fat. The fatty livers of the Strasburg geese are produced in this way. (2.) When, in the course of an exhausting disease, rapid emaciation takes place, and causes the blood to become loaded with oil from the waste of the adipose tissue. (3.) When the type of respiration is low, and the blood necessarily, therefore, contains much hydro-carbonaceous matter. In the first and third classes there may be simply accumulation of a large quantity of oil in the tissue of the liver; in the second there is generally, also, more or less degeneration of the hepatic structure. We have verified this by positive observation, as respects the first and second classes. Animals fed for some time with fatty food have their livers loaded with oil, but the cells are not at all destroyed; they contain much oil, and much also is deposited between them. We have found the liver, in persons dying in a condition of extreme emaciation from other diseases besides pulmonary phthisis, in a complete state of fatty degeneration; and this has occurred so often, that we should expect to find it in most cases of great general wasting. It is certainly the emaciation of consumptive disease that produces fatty transformation of the liver, and not the mere destruction of the oxygenating apparatus. This statement is strongly confirmed by the analysis of about a hundred cases, in which we examined, microscopically, the condition of the liver. Among them there were eighteen in whom the liver was thoroughly fatty, or nearly so; of them, only *one* died of pulmonary phthisis, four others of scrofulous affections, the remainder of diseases having no connection with any form of phthisis: ten cases of phthisis occurring in the same list presented either no fatty degeneration, or an imperfect and partial change only. A fatty state of the liver is sometimes coincident with a similar condition of the kidneys, but not by any means invariably. In well-fed persons living in towns, some degree of fatty change in the liver seems to be the rule. It is also a consequence of excessive drinking; especially, it is said, of drinking dilute forms of alcohol, as beer, but we have also seen it in spirit drinkers.

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* Authors have drawn a distinction between fatty infiltration and fatty degeneration: the former being analogous to the normal or physiological deposition of fat in the liver cells; the latter an actual conversion of their substance into fat. It has further been said that the latter is distinguished by exhibiting the fat in a finely granular form. We have been unable to trace this distinction in the individual cell, and believe the difference, if there be any, to exist rather in the general course of the process.

(2.) *Granular or Parenchymatous Degeneration.*—In many acute febrile diseases, some swelling of the liver is noticed during life. When this is the case, we find the liver enlarged, either pale or vascular and opaque, somewhat resembling a fatty liver; so much so, that it may sometimes be a matter of doubt whether there is a chronic fatty change, or an acute degeneration. The substance is, however, more flabby than in the fatty liver, the colour less yellow; and, in advanced cases, the lobular structure much less distinct. Microscopical examination shows the cells punctated with highly refracting granules, which in early stages are soluble in acetic acid or alkalies, and are probably albuminous in composition. The cells are at the same time swollen. This condition has been called by Virchow cloudy swelling. In a more advanced stage the granules are insoluble in acetic acid, and are in fact fatty, so that the condition ends in fatty degeneration. A similar simultaneous change is often seen in the renal epithelium. This form of degeneration occurs in erysipelas, diphtheria, pyæmia, and other specific fevers; also in diseases such as pneumonia, accompanied by fever. It has been explained simply as an effect of high temperature.

(3.) *Lardaceous, or Waxy* (*amyloid*) *Degeneration* of the liver, presents the same general features as in other organs, and here, too, is said to commence in the smaller arteries or capillaries; but even if this be the case, the most prominent and characteristic alterations are seen in the hepatic cells. These are found enlarged, rounded, and indistinct in outline, translucent or hyaline in substance, the nucleus no longer perceptible. The individual cells become after a time fused together into irregular masses. The change always begins in that part of each acinus which immediately depends upon the hepatic artery and portal vein, that is, in the peripheral portion; while the central portion, or that connected with the hepatic vein, remains long unaffected. The small arterial branches become blocked, as is shown by the difficulty of injecting the organ through the artery.

The liver thus affected is almost always enlarged, sometimes very greatly; but this, perhaps, is partly dependent upon the fatty degeneration which almost always accompanies the lardaceous change. The edges are rounded, the substance extremely firm, tough, pale, and translucent. As the change seldom extends quite uniformly through the whole organ, the translucent portions are mottled with opaque white fatty patches. In rare cases, the lardaceous change is said to occur only in isolated spots. Iodine produces in the liver thus affected very characteristic markings, the degenerated portions being stained orange, or dark brown; and, from the peculiar distribution of the change, the acini are very clearly defined. Treated with sulphuric acid, the iodine-stained portions become nearly black.

(4.) *Pigmentary Degeneration* of the liver is described as the consequence of intermittent fevers, which produce a similar affection of the spleen. This condition is seldom seen in this country. It is described by Klebs as arising from deposition of black pigment in the portal capillaries, and in the interlobular branches of this vessel; later on in the hepatic capillaries also. The liver is at once distinguished by its dark, greyish brown, or nearly black colour; which is either general, or limited to the territory of the portal vein. The size is at first increased, but in the end atrophy always sets in, which may be considerable. The pigmentary matter consists of round or angular granules of a blackish colour, and is always contained within the vessels, either free, or enclosed in cells, some resembling ordinary leucocytes, some angular, like the epithelium of the splenic vessels. The source of this pigment is believed to be the spleen, where a similar change is constantly found in cases of intermittent fever, and there depends upon stagnation of blood in the sinuses, and the metamorphosis of its colouring matter. The obliteration of the vascular channels leads to congestion of the portal venous system and further consequences, which are nearly the same as those of cirrhosis. Ultimately the pigment may find its way through the liver into the hepatic venous system, and thus become distributed over the body.

Simple Atrophy of the Liver.—Wasting of the liver occurs in many diseases, accompanied by general marasmus, unless (as in phthisis) fatty degeneration supervene, and is very noticeable in cases of starvation. It also results from any obstruction of the portal vein. In some cases an idiopathic atrophy appears to occur without any obvious cause. The organ is much reduced in size, somewhat granular on the surface, of firm but not hard consistence, and of a peculiar brownish red colour, with the lobules clearly defined, so that it has some resemblance to the liver of chronic venous congestion. It has, on the other hand, often been described as a form of cirrhosis; and it is probable that extreme wasting from the latter cause may come to resemble very closely simple atrophy. The distinction of course depends on the absence of any new production of fibrous tissue in the stroma, *i.e.* of interstitial proliferation, in the cases of simple atrophy. The fibrous tissue and Glisson's capsule may, however, be apparently increased in consequence of the wasting of the hepatic tissue. Dr. Wilks describes a case in which the organ was reduced to $1\frac{3}{4}$lbs. in weight, and the liver-cells were not more than one-half or one-third of their normal dimensions. He characterizes such livers as being smooth on the surface, and homogeneous in substance, but as producing the same obstruction of the portal system as actual cirrhosis.

THROMBOSIS AND INFLAMMATION OF THE PORTAL VEIN.

Coagulation of blood in the portal trunk or some of its branches, accompanied or not by inflammation of the wall of the vein, may be produced by several causes, and lead to important changes in the liver.

This condition in the portal or hepatic vein branches has already been alluded to as resulting occasionally from abscess of the liver, but it may be produced by other causes. A fish-bone has been known to perforate the wall of the stomach, and the head of the pancreas, and wound the superior mesenteric vein, exciting thus inflammation of the coats of the vessel which extended to the divisions of the portal vein. The effects of thrombosis and phlebitis are the same in these as in other veins; they may simply occasion the blocking up of the channel with fibrinous coagula, or these may proceed in some portions of the vein to puriform softening. Sometimes we find the wall thickened and inflamed with the coagula adherent to it, sometimes quite unchanged. In some cases a sort of embolism appears to take place within the portal system; portions of clot being transferred from the tributary veins of the portal trunk to some of its branches. This is especially the case when the coagula soften. A remarkable case of this kind is described at page 404.

If simple obliteration occur the clot becomes inseparably united with the inner coats of the venous wall, probably through growth from the latter.*

When the inflammation has subsided, the obstructed vessel gradually shrinks, and is reduced to a fibrous cord, while the surrounding tissue to which it was distributed, being deprived of its supply of blood, atrophies and falls in, so that the course of the vein is indicated by a deep linear fissure. The consequence of such attacks must be the diminution, to a greater or less extent, of efficient hepatic parenchyma, and as the importance of this structure to the due performance of nutritive absorption is very manifest (were it attested only by the intimate relation in which it is placed with the portal blood), it seems very probable that the abiding emaciated and enfeebled condition of many persons who have suffered inflammatory attacks of the liver, really depends on their having been thus deprived of a greater or less part of this important organ.

Partial obstruction of the portal trunk produces general wasting of the liver. Total obstruction is fatal. The spleen always becomes enlarged; in the case above referred to it weighed twenty-four ounces.

Breaking down, or puriform softening of the thrombus, may produce abscess in the liver, but does not always do so. Perhaps, as in other instances of softening clots, this is only the case when the special poisoning of pyæmia is present, and is conveyed to the organ by the softening clots.

CONDITIONS PRODUCING JAUNDICE.

The next morbid condition of the liver which we shall consider is that which exists in the various affections in which *jaundice* forms a prominent symptom. There is little doubt that in most cases a jaundiced condition of the liver precedes and occasions a similar condition of the whole body. General jaundice is commonly supposed to depend upon the absorption into the blood of bile that should have passed out into the intestine, and this is, doubtless, the cause of it in many cases. In these, the bile locked up in the substance of the liver causes it to be tinged yellow, a result which we have produced artificially by placing a ligature on the duct. com. choled. in animals. But in many cases more than this occurs. Dilatation of the heart, or obstructive valvular disease, throwing back the blood on the venous system, occasions permanent congestion of the liver, and often produces the condition termed "nutmeg" in its most marked form. In this, as before described, the congestion is exactly co-extensive with extreme yellow engorgement of the cells; a much larger quantity of yellow matter is contained in the liver; there is hepatic jaundice, and together with this, and, no doubt, in consequence of it, general jaundice frequently occurs. In these cases we think the evacuations continue of their natural colour; a certain quantity at least of bile flows into the intestine. In most healthy animals the cells of the liver have only a very faint, if any, yellow tinge, but by repeated doses of calomel we have caused the production of a large quantity of yellow matter in the cells; there is evidence also to show that the same has occurred in the human subject. In the acute yellow atrophy, as Rokitansky names that condition of the liver (of which we shall speak presently), attended with symptoms of toxæmia, and proving fatal by coma, there must certainly be a greatly-increased production of yellow pigment. The flow of bile into the intestine is not so completely stopped as it is in other instances of jaundice, and the yellow coloration of the liver is deeper than it is almost ever seen.

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* See a case in "Trans. Path. Soc.," vol. xxiv. p. 122.

Icterus neonatorum appears also to be an instance of the excessive production of bile or of bile-pigment: there is evidently no disease of the liver, or any obstruction in the biliary ducts, but owing to the organ at birth being highly congested with blood, and the system not having adapted itself to its new condition, a greater quantity of yellow pigment is formed out of the colouring matter of the blood than can be readily carried off by the bile; this again returns into the blood and produces jaundice. This explanation seems preferable to that which assigns a kind of hyperæmic or half-bruised state of the skin, as the cause of the yellow stain; were this so, how could the conjunctivæ come to be affected? When jaundice occurs in the course of fevers or in pyæmia, it then depends, in all probability, on an alteration taking place directly in the hæmoglobin of the blood, which, as in the case of an extravasation, is changed from a red to a yellowish tint; in this case there would be no preceding jaundice of the liver. It is evident that in all instances of jaundice the unnatural tint results from the presence of a yellow (usually identical with bile) pigment in the blood: this is easily demonstrable in the urine and other secretions by the stain imparted to linen, or by the play of colours which it gives with nitric acid; but there is little or no evidence to show that real *biliary matter*, *i.e.*, the bile acids, are present in the blood or the secretions. From jaundiced livers plenty of yellow pigment can be extracted, reacting with the nitric acid test, but no cholic acid, or any substance that gives the reaction of Pettenkofer's test. The blood, in cases of jaundice, is more often found to contain bile-pigment without cholic acid, than the reverse. The same is the case with the urine; it often gives a characteristic reaction with nitric acid when none is afforded by the test of sugar and sulphuric acid. From these data we must conclude that jaundice depends on the presence of bile-pigment or some similar modification of hæmatine in the blood; but that it does not follow that bile, as such, is in all cases actually present. It may be that, in the graver cases of jaundice, attended with toxæmia, cholic acid, or some modification of it, is present in the circulation in large quantity, as well as yellow pigment. The colour of the liver in jaundice is of a more or less marked yellow, in some cases passing to a green or brownish tint; this will be, of course, modified and more or less concealed by the blood contained in the vessels. Jaundice may also be produced by changes in the bile ducts, which will be subsequently spoken of.

ACUTE YELLOW ATROPHY.

This rare and remarkable affection seems to differ very much from the other morbid conditions producing jaundice. The size of the organ is here very greatly diminished, being sometimes less than 2 lbs. in an adult man. The colour is usually an intense yellow, but sometimes orange-coloured or decidedly red, its texture is flabby, a section shows nothing of the natural lobular arrangement, and it seems almost or quite bloodless. The blood in the large vessels is said by Rokitansky to be reduced in consistence, and of a dirty reddish-brown colour, and the coats of the V. Portæ to be tinged with bile. Under the microscope it is seen that the cells are completely destroyed, and even their nuclei have perished, the parenchyma is a mere mass of broken-up granular matter, tinged deeply yellow and containing some largish yellow masses, together with diffused oily matter. In one instance which we examined, no sugar could be extracted from the liver, though it is usually abundant in healthy organs: this may, however, have depended on the non-ingestion of food for some time before death. The minute ducts we have found gravely altered; they had lost their natural structure, and were filled with subgranular matter and opaque whitish globules. These globules, probably leucin, were very abundant in the lymphatics, and rendered their course remarkably distinct. Several observers describe the bile ducts as empty or containing only mucus, and never bile.

Two chemical substances are very constantly found in the broken-down hepatic tissue, viz., *leucin*, which occurs in whitish opaque globules, sometimes concentrically stratified, or in aggregations of such globules, and *tyrosin*, which is found in bundles of acicular crystals. Both are nitrogenous substances, probably derived from the decomposition of albuminous matters. They are also found in the urine, when the liver is affected with this disease, and normally in the spleen, pancreas, and some other parts of the body.

Some pathologists have described a condition of *red atrophy*, very similar to the yellow, and occurring likewise in an acute form, with febrile symptoms; but we do not see sufficient reason for making it a distinct disease. The name is also unfortunate, as it has been used for the very different condition of "nutmeg" liver.*

Partial acute atrophy sometimes occurs, the characteristic changes being found in certain portions of the liver, while the rest is normal.

Causes of Acute Yellow Atrophy.—The causes of this disease are unknown. It has been found that phosphorus poisoning produces a somewhat similar affection, and hence it has been suggested that there may be cases of accidental phosphorus poisoning, which seems very unlikely. Syphilis has been present in a rather notable proportion of cases, but can hardly be invoked as a cause. Some observers have referred the whole to acute inflammation.

TUMOURS AND NEW GROWTHS OF THE LIVER.

Simple tissue tumours are not common in the liver. The following have been observed as primary growths. (1) Lipoma; (2) Fibroma; (3) Angioma, or cavernous tumour; (4) Lymphoma.

(1) and (2) are extremely rare, and of no practical importance.

(3) Cavernous tumours of small size are not very uncommon, though it would seem less common in this country than in Germany. In the ordinary form a bluish patch is seen on the surface of the liver, which when cut into is found to represent a cavernous or spongy structure of intercommunicating spaces occupying the place of liver tissue, and usually containing liquid blood, but sometimes firm thrombi. They may also occur at a distance from the surface. The size varies from that of a pin's head to masses some inches in diameter. These singular structures have been explained by Virchow as formed by the metamorphosis of the capillary network of the liver, and having no special connection with any one of the chief systems of vessels. This is not, however, always the case, as appears from the following description of one of the most remarkable instances on record, in which large and numerous cavernous tumours, containing coagulated blood, were found scattered through the liver.

The system of sinuses composing the cavernous tissue was found to stand in immediate relation with the portal veins. The two main divisions of the portal vein supplying the left lobe opened into two of the largest cavernous tumours by channels as large as a goose-quill, and nearly all the larger branches of the portal vein were found to be similarly connected. On the other hand, the larger branches of the hepatic vein were quite unaffected, and no communications, but small and inconspicuous ones, could be traced with this system of veins, nor any with the hepatic artery. The smaller tumours were enclosed in a capsule and easily separable from the surrounding tissue, being attached to small branches of the portal vein like fruits to their stalk. The larger tumours had no capsule, and were not separable from the liver tissue.*

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* Dr. Andrew describes in "Trans. Path. Soc.," vol. xvii. p. 158, a case of acute atrophy in which the liver had an orange-red or red colour.

Lymphoma or Lymphatic Tumours.—In cases of leuchæmia, the liver is often greatly enlarged, and contains numerous masses of lymphatic elements, such as have been already stated to occur in many organs in this disease. These tumours are distinguished by their white colour, soft and somewhat crumbling consistency. They are often extremely numerous and small, but sometimes form larger masses; and sometimes there is a more general infiltration. The microscopical structure is little more than a collection of lymph corpuscles, with very little fibrous stroma or intercellular substance.

Lymphadenoma.—In certain cases of *anœmia lymphatica*, or Hodgkin's disease (p. 425), growths and infiltrations of cytogenous tissue are met with, which are usually firmer than the purely leuchæmic tumours, from the presence of more intercellular substance; and in the forms called lymphosarcoma, by Virchow, a greater variety of cell forms is sometimes observed.† The latter tumours are yellowish, translucent, and of firm, tough consistency.

Simple Cysts (unconnected with hydatids) have, in a few instances, been found. Dr. Bristowe has described cases of very numerous cysts in the liver, associated with a similar cystic condition of the kidneys,‡ and Dr. Wilks speaks of similar cases.

Sarcomatous tumours occur not very unfrequently as secondary growths. Spindle-celled sarcoma and melanotic sarcoma have been thus observed.§ One case of primary melanotic sarcoma in the liver is recorded by Frerichs. One case of secondary myxoma has been recorded,|| and secondary enchondroma has also been observed.

Cancer is very frequent in the liver: it stands fourth in the list of organs thus affected, according to the Parisian registers; these show that it occurs about once in every sixteen cases of cancer. Rokitansky estimates its occurrence in the liver to be much more frequent; he states "its numerical relation to carcinoma of other organs, as 1 to 5." The above statements do not, of course, refer to primary cancer of the liver only; but include secondary cancer also. Three varieties of cancer, beside epithelioma, have been observed in the liver; colloid is extremely rare, neither Dr.

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* Payne, "Trans. Path. Soc.," vol. xx. p. 203.

† Payne, "Trans Path. Soc.," vol. xix. p. 401; Murchison, Ibid., vol. xx. pp. 192 and 198, pl. vii. and viii.; Wilks, "Guy's Hospital Reports," 1856, series 3, vol. ii. p. 114.

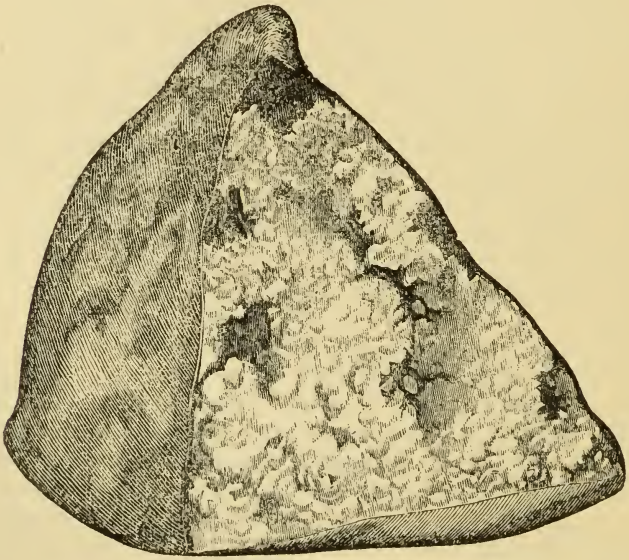
‡ "Trans. Path. Soc.," vol. x. p. 174.

§ Murchison, "Trans. Path. Soc.," vol. xxiv. p. 123; A. Clark, Ibid., vol. xxiii. p. 251; vol. xxiv. p. 134.

|| "Trans. Path. Soc.," vol. xxiv. p. 120.

Budd nor Dr. Walshe has met with it; Rokitansky seems only to have seen a single case, and he does not state whether it was primary or secondary. Scirrhus is not very unfrequent, or a transition variety between it and encephaloid. The commonest form of cancer in the liver has an intermediate hardness, greater than that of ordinary encephaloid, but inferior to the stony hardness of scirrhus of the mamma; it constitutes roundish tumours, about the size of a large nut, whitish, fibrous, and tolerably firm. Encephaloid is far the most common, and, as in other parts, attains far the largest size. We have seen almost the whole organ converted into a mass of this kind. It sometimes forms separate tumours, sometimes infiltrates the parenchyma. Rokitansky's description of the separate tumours seems to us to apply equally to the scirrhous and encephaloid varieties, as he himself appears to allow. He says, "Their general form is spherical, though their surface not unfrequently is slightly racemose or lobulated. Those which have been developed in the peripheral portion of the organ, and are, therefore, in contact with the peritoneum, present a flattened or even a puckered surface, being drawn in at the centre or umbilicated, and the contraction may extend to the very nucleus of the morbid growth. The peritoneal covering over the contracted portion is opaque and thickened," probably from having become involved in the cancerous degeneration; it seems to be retracted and drawn in much the same way as the skin is in subcutaneous cancer. The number of the cancerous

Fig. 143.



Encephaloid growth, occupying a large extent of the liver.

tumours varies in different cases; they may be solitary or very numerous; primary cancers are usually few, secondary may amount to some hundreds. In rare cases minute tumours are distributed so generally through the organ as to produce the effect of an infiltration. Dr. Walshe thinks they are most numerous when they occur consecutively to cancer of the stomach. The scirrhous tumours have scarce any investments of cellular tissue, and adhere closely to the surrounding hepatic parenchyma; the encephaloid have a delicate cyst-like investment, though this does not seem to be constant, and they can be detached more readily. "Infiltrated encephaloid," according to Rokitansky, "always contains obliterated and obsolete blood-vessels, and ducts which are gradually absorbed. The infiltration attacks larger or smaller segments of the viscus, it does not present distinct boundaries, but insensibly passes into the normal parenchyma. It rarely occurs without nodulated cancer." The separate tumours often enclose strata of remaining hepatic structure, a fact which seems to mark a connection between the two forms; some degree of infiltration taking place in each; but in one, the growth simply pushes the parenchyma aside, in the other it spreads its germs everywhere among its elements. The structure of cancerous tumours presents nothing different from that of cancerous tumours in other parts, and is described under the general head of cancer (p. 172). Their degree of vascularity varies: some tumours show very little trace of blood-vessels, others are richly supplied, and are the seat also of interstitial effusions of blood; to such the term hæmatoid or fungus hæmatodes is appropriate. Black pigment is often scattered through the substance of the growths, and may be so abundant as to make them appear entirely black. These claim, of course, the appellation melanotic, but most, if not all such, are cases of sarcoma.

Cancerous tumours in most cases produce considerable enlargement of the liver, and the very largest livers known are produced by this cause. The largest we have seen weighed more than 16 lbs., but still larger are on record, the atrophy of the proper tissue which they occasion being more than compensated by the amount of their own enlargement; in some rare cases, however, this does not take place, and the liver, though containing many cancerous tumours, is smaller than natural. Masses of cancer which appear on the surface of the liver sometimes excite adhesive inflammation of the investing serous membrane, and thus become united by false membrane to adjacent parts. Instead of this, they have been known to infect with their tainted fluids the parts in contact with them, and to cause secondary formations of cancer in them, or to extend into them, by the ordinary way of infiltration. Ascites, to some extent, is not unfrequently produced by the presence of cancerous masses in the liver: this probably depends on the obstruction of the portal vein branches, either by the tumours themselves, or by cancerous matter developing in them, or by fibrinous masses coagulated within their channel. Jaundice is often observed in cancerous disease of the liver: its production, doubtless, takes place in the same way as that just noticed; the gall ducts being obstructed, and the escape of bile from various parts of the organ prevented. When the masses are so situated as to press on the common duct leaving the others free, enormous distension of the gall-bladder may take place—it has been seen as large as the fœtal head; such a result, however, is more likely to be produced by cancerous disease of the head of the pancreas, than by growths in the substance of the liver. Primary cancer of the liver is stated by Dr. Budd, seldom, if ever, to occur before the age of thirty-five: from this to fifty-five is the epoch at which it most frequently manifests itself; but it is very rare at any age, so much so that some observers have called in question the occurrence of primary cancer in this organ.

Secondary cancers of the liver may occur at any age: they seem, according to Dr. Walshe, to affect a preference for the superficial parts of the organ. They are believed to be produced by the transportation of germs in the blood, or the lymphatic vessels.

The former mode of transmission is sometimes very clearly shown by the occurrence of growing masses of cancer within the smaller branches of the portal vein, starting from which they infiltrate the organ. Such growths are most often seen after primary cancer of the stomach or intestines. Infection by the lymphatics is seen in cases where morbid growth creeps up into the liver by the tissue surrounding the portal vein and hepatic artery; and in some cases cancer of the pylorus, duodenum, or head of the pancreas invades the liver by direct contiguity. Nevertheless, in most cases of secondary cancer the mode of conveyance of germs cannot be traced; it may, however, occur in the liver, subsequent to disease of the mamma, kidney, uterus, or any other part. The secondary growth has been asserted by Waldeyer to take place solely from the conveyed cells or germs; but it is more probable that the liver structures participate in the new growth, and probably the connective tissue only, though according to some observers the liver cells also are concerned.

Epithelioma.—The flat-celled epithelioma occurs of course only as a secondary growth, and then very rarely. Cylindrical epithelioma may occur secondarily to similar growths in the intestines, but also in a very remarkable form of primary tumour.

Cylindrical Epithelioma, Adenoma of the Liver—This remarkable form of tumour has at present been observed in very few instances. In one which has fallen under the observation of the editor numerous tumours were found scattered through the liver, which was greatly enlarged. The tumours were firm, white, moist, and fairly vascular; they showed on the surface central depression or umbilication, but otherwise no sign of degeneration. No juice was obtained by scraping, but in general their appearance was precisely that of an hepatic cancer of medium firmness between scirrhus and encephaloid. The liver growths were plainly primary, no others occurring in the abdominal viscera, but there were secondary nodules in the lungs. The minute structure of the tumours had the most striking resemblance to that of a secreting gland, consisting of tubes and cæcal pouches, lined with a perfectly regular epithelium, usually cylindrical, sometimes spheroidal, or even resembling a pavement epithelium. There were also cystic spaces filled with clear fluid.*

SYPHILITIC AFFECTIONS OF THE LIVER.

Syphilis produces several distinct morbid processes in the liver.

1. *Minute granulations* are sometimes found in newborn children affected with congenital syphilis, and were first noticed by Gubler. They are described as varying from the size of a pin's head to a pea, of yellow crumbling consistence from early fatty degeneration; and thus with much resemblance to miliary tubercles.

2. *General interstitial hepatitis*, or cirrhosis, appears to be sometimes a consequence of syphilis, cases occurring in syphilitic individuals in whom no other cause of cirrhosis can be demonstrated. It is possible that the rare cases of cirrhosis in young children, already referred to, are due to inheritance of this disease. The perihepatitis which often accompanies this condition has been already spoken of.

3. *Gummata*, or syphilitic tumours, are the most frequent and characteristic products of syphilis affecting the liver; and have long been known, though they were formerly regarded as healed cancers. They agree in general with the description already given (p. 203) of syphilitic gummata, but have in the liver certain peculiarities. The first stage is rarely met with, but has, under peculiar circumstances, come under the observation of Messrs. Cornil and Ranvier, who describe the gummata in this stage as highly vascular, of a pinkish grey colour, without any juice, and of firm consistence. The minute structure is made up of embryonic cells, with intercellular substance, showing a decided tendency to necrosis and degeneration into a molecular detritus. In the margins more distinctly formed connective tissue is seen.

The second stage, which is marked by retrogressive changes, is that most frequently seen. Masses of tough, firm, opaque, yellowish material are seen imbedded in a liver which is usually otherwise normal, and are either surrounded by a more or less distinct fibrous capsule, or else pass by an imperceptible transition into the surrounding tissue. They are distinguished from tubercular masses by the size they often attain, and from cancer or any form of tumour by the peculiar consistency. The microscopical appearances of this stage are in the main what have been already described. There is much less difference than might be supposed between the yellow and the vascular portions, both consisting of adenoid tissue with nuclei.

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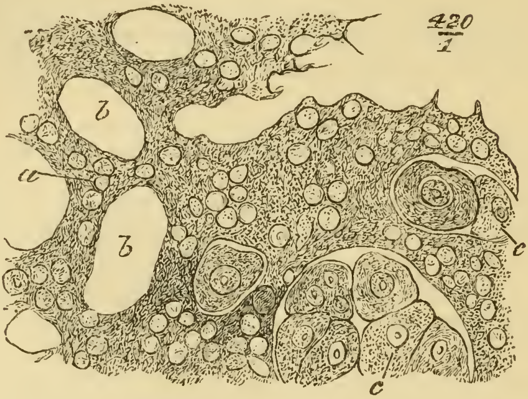
* The case is reported by Dr. Greenfield, "Trans. Path. Soc.," vol. xxv.

FIG. 144.



Section of liver showing the mode of growth of syphilitic tumours.

a, Nuclei of the new growth. *b*, Spaces from which liver cells have dropped out. *c*, Remaining liver cells.

The growth, or at least the increase of these tumours, is effected by a gradual infiltration of the liver tissue with a nucleated new growth, while the liver cells appear to give way or become absorbed. In Fig. 144, which represents the growing margin of a syphilitic tumour of the liver, we see islands of liver tissue gradually compressed and destroyed by the growth of a tissue composed of nuclei imbedded in a firm fibrous matrix; corresponding precisely to the type of syphiloma. This is clearly the method in which such tumours increase, and by analogy we may suppose that it was the method in which they originated, though, as in many similar instances, this may be open to some doubt.

In the third stage the central portions become more crumbling and cheesy, while the production of fibrous tissue in the peripheral parts forms a distinct capsule, so that an almost cystic structure results. The central part is rarely found in an actually softened condition, but we have seen one case in which pultaceous semi-fluid matter could be pressed out, precisely as from some tubercular vomicæ in the lung. By some such process the degenerated portions often become completely absorbed, and nothing but a mass of fibrous tissue may remain. This is distinguished by its remarkable power of contraction, in consequence of which the liver tissue of the part affected being already destroyed, remarkable changes of form are produced in the liver. Puckered scars are seen on the surface, which sometimes penetrate very deeply, and may almost cut off one part of the organ from the rest. In the end it seems that complete absorption is possible, and nothing but a scar may remain. These scars are themselves very characteristic, and a puckered depression on the surface of the liver, especially if underneath it is found a small mass of fibrous or cheesy matter, may be regarded as almost certainly syphilitic. The organ may, if these scars are numerous, assume a peculiar lobate appearance, like the compound livers of some of the lower animals. More rarely calcareous metamorphosis of the degenerated tissue occurs.

Tubercle rarely occurs in the liver, except in cases of acute general tuberculosis. In this disease miliary tubercles are occasionally seen scattered through the gland, and more frequently still the microscope reveals minute tubercles, not visible to the naked eye. They are collections of small round cells, but are distinguished from simple interstitial proliferation by their definite form, and by the central degeneration. Also in cases of tubercular disease in children, not precisely acute, we sometimes find evidence of tubercular disease in cheesy or calcareous nodules rarely larger than a pea, often bile stained, and asserted by some authorities to be specially connected with the bile ducts. The tubercle seldom, if ever, proceeds to an extreme degeneration or softening, so as to produce anything like an hepatic vomica.

PARASITES IN THE LIVER.

Hydatid, or *echinococcus cysts* are of frequent occurrence in the liver, more so in this than in any other organ. Sometimes they are single, sometimes there are several separate cysts. They often attain a considerable size. Rokitansky mentions one in the Vienna Museum of a foot in diameter, and we have very recently examined one of an oval form, whose long diameter measured six inches. Their usual site is the right lobe, and the largest are generally found here; but the one just mentioned was situated at the extremity of the left, and had grown in, and far beyond, the left lateral ligament. As their size increases they rise to the surface of the liver, and sometimes excite inflammation of the serous membrane, by which adhesions are formed connecting them with the parts adjacent. The prominent part is, of course, that where least resistance is offered to the pressure of the fluid within, and its wall may hence atrophy and give way, or be destroyed in the course of suppurative inflammation, and the contents thus be effused into some neighbouring cavity. The cysts have been known to burst into the peritoneal sac, into that of the right pleura, or into the bronchi of the corresponding lung, into the duodenum or transverse colon, and in some rare instances, into a large blood-vessel or branch of the hepatic duct. When the tumour, in its progress, causes ulceration of one of the smaller ducts, which is not uncommon, bile makes its way into the cavity, mingles with and tinges its contents, and very often excites suppurative inflammation of the walls of the sac. This seems to be the reason that hydatid tumours in the liver suppurate much more frequently than those in other parts.* Other circumstances, however, may certainly cause these cysts to inflame and suppurate. The detailed description of the structure of hydatid cysts will be found under the head of Parasites, p. 218; it will, therefore, be sufficient to mention here that they possess an outer wall or envelope, formed of condensed areolar tissue and that of the surrounding structure; within which, and rather loosely adhering to it, is the proper membrane. This is white and laminated, and is itself lined internally by a softish layer in which the echinococci are developed. The cavity of the primary cyst is occupied in some instances by a transparent limpid fluid only; in others, and the majority, it also contains a numerous progeny of secondary cysts, sometimes sterile, sometimes containing innumerable scolices or echinococcus heads, known under the microscope by their ring of hooklets. Dr. Budd mentions the interesting fact, that in cases where suppuration has occurred in the cavity of the primary cysts, the secondary hydatids, though floating in purulent matter themselves, contain a perfectly limpid fluid. He also points out characters whereby to distinguish between an abscess and a suppurated hydatid cyst, in the differences which the cystic membranes in the two cases present. That of an abscess consists of dense fibroid tissue, is not laminated, and never contains calcareous matter. The hydatid membrane does not adhere so firmly to the surrounding tissue, is markedly laminated, and in old cases contains very often plates or grains of calcareous matter in its coats. When an hydatid tumour has evacuated its contents as above described, it may collapse and a cure be effected; but if its walls are very thick and firm, and the cavity large, its obliteration in this way may be impossible, and then there is too much reason to fear that, owing to the entrance of air or other matters, suppurative inflammation of the sac will be excited, and the drain exhaust the strength of the patient. But an hydatid cyst may come to a spontaneous cure in a different way; its proper membrane, instead of secreting a watery fluid, may produce a putty-like matter, consisting of phosphate and some carbonate of lime, with cholesterine and albuminoid matter, generally stained with bile. This accumulates within the sac, or sometimes around it, imbeds the secondary hydatids, and causes them to shrivel up and perish. Such a change reminds one forcibly of the cretification of tubercle, which is often observed in cases where the tubercular process has ceased, and the deposited matter has been partially absorbed. Hydatids in the liver are not unfrequently associated with hydatids in other parts, in the lower lobes of the lungs, or in the spleen, or in the mesentery; in such cases Dr. Budd is inclined to regard the hepatic cyst as the parent, and the others as originated from germs conveyed from it; but there is no reason to think that such a transference can take place within the same body, and it is more likely that several parasites or ova originally entered the body simultaneously. The peculiar form called echinococcus multilocularis has already been mentioned (p. 219). It has never been seen in this country, and is confined, almost without exception, to South Germany and Switzerland.

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* Some doubt may exist whether the purulent-looking fluid contained in the cyst is always true pus. In a case occurring at St. Mary's Hospital, the matter from the interior of a large cyst, which had to the naked eye all the appearance of pus, was found under the microscope to consist of much granular and oily matter, with some cholesterine, and numerous utterly irregular granular masses. There were no true pus globules.

Distoma hepaticum, or the fluke, has been found in the human liver, and the ordinary parasite of the rabbit's liver, the *Pentastomum denticulatum*, occurs very rarely; usually encapsuled and calcified.

ABNORMAL CONDITIONS OF THE BILIARY PASSAGES.

Malformations.—The gall-bladder is sometimes wanting—in animals it has been found double; its shape may be variously deformed; its duct, as well as the common duct, may probably be imperforate. The cystic and hepatic duct may remain separate, and communicate either both with the duodenum, or one with the duodenum and the other with the stomach.

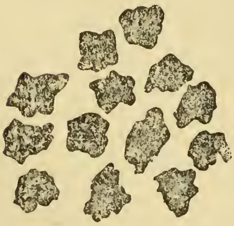
Inflammation.—The mucous lining of the gall-bladder and ducts is often attacked with inflammation, which may extend from the duodenum, and spread upwards along the ducts. It is often of the catarrhal kind, and is essentially similar to the affection of the gastro-intestinal mucous surface; like it, subsiding after a time, and leaving no traces of its existence behind. The effects it produces will be those of vascular injection, some degree of tumefaction, shedding to a greater or less extent of the epithelium, and casting off of mucous corpuscles and various forms of immature epithelia, together with exudation of *liquor muci* of various degrees of viscidity and tenacity. The gall-bladder alone may be the seat of acute idiopathic inflammation, or this may be excited by unhealthy bile, or, perhaps, by the irritation of a calculus. The result of such inflammation may be closure of the cystic duct, and conversion of the gall-bladder into an abscess. If the catarrhal inflammation, or that set up in any other way, attain a certain degree of intensity, it causes the effusion of muco-purulent or purulent matter, and at the same time it seems to induce paralysis of the contractile coat of the biliary ducts; these tubes thus weakened, yield to the distending force within of the accumulating secretion, and become dilated at intervals into cyst-like pouches, filled with muco-pus, tinged yellow or green by bile. The dilatation will, of course, be promoted, if the common duct, or the hepatic, is obstructed by a calculus, or in any other way. After such pouches have existed a certain time, they become entirely cut off from the duct in which they originated, the tube becoming obliterated by adhesion, and their contents then undergoing certain changes. Thus the muco-purulent matter may be converted into a clear glairy fluid, more or less tinged with bile; we have recently observed a case of this kind, and though we were some time in doubt as to the nature of the cyst, which was found in a healthy liver, we were soon convinced by detecting particles of columnar epithelium in the matter lining its surface; besides the fluid in this case, there were several small whitish masses attached to the inner surface, consisting of a semi-homogeneous, semi-granulous, soft substance, containing imperfect celloid forms. We think it is too much to assume that all such tumours as contain a glairy fluid have originated in catarrhal inflammation; probably the morbid cyst-producing action is in many cases of a more chronic kind, and the fluid is glairy from the first. In a boy who died with pneumonia supervening on a tuberculized state of the lungs, we found the liver, with the exception of marginal oily accumulation in the lobules and a somewhat atrophied condition of the cells, apparently healthy, except that here and there throughout its substance there were seen green coloured spots of the size of a pin's head. These seemed to exist about the termination of the minuter portal canals, and were, doubtless, connected with the terminal ducts; they consisted of yellow and orange or reddish pigment colouring matter, heaped up together and forming a mass which encroached on the parenchyma; in one of them columnar epithelial particles were seen, proving that a duct was involved in it. The cells contained no yellow matter, so that it was evident that these green masses had been produced by a morbid action set up in the minute ducts. Ulceration of the gall-bladder is not unfrequent; it may occur as a consequence of suppurative inflammation, or be set up in an organ which has been the seat of chronic disease, or occur in the course of remittent or typhoid fever, or be produced by the irritation of calculi, and probably also by that of unhealthy and acrid bile. The ulcers are sometimes small and numerous, sometimes there is but one large one: they are sometimes attended with sloughing of the coats, and sometimes go on to perforate the wall completely. When this happens the bile, if the gall-bladder contain any, escapes into the cavity of the peritoneum and rapidly excites fatal inflammation. If from long closure of the cystic duct, the gall-bladder contains no bile, but only a mucous or serous fluid, this does not escape so rapidly, and the inflammation is more limited to the neighbourhood of the liver. When ulceration is excited by the presence of gall-stones, it usually happens that the bladder becomes adherent to some adjacent part, commonly the colon or duodenum, and, as the process advances, a communication is established between the two viscera, by means of which the calculus escapes into the bowel and may be discharged. We have, however seen a case in which fatal obstruction of the intestines was occasioned by a gall-stone, which had probably escaped by a fistulous opening from the gall-bladder. We do not know much of ulceration of the ducts; the smaller branches are so rarely examined, that its existence may have been overlooked. Dr. Budd records one very interesting case in which an ulcer of the common duct made its way into the superior mesenteric vein, close to its termination in the portal; the result was phlebitis of the vessel and purulent formations in various parts of the liver and the lungs, as well as in the skin and subcutaneous tissue of the head and face, and in some other parts also. If inflammation, attended, probably, with some amount of ulceration, attack the cystic duct, obliteration of its channel may be the result, or the same may be occasioned by a gall-stone lodging there. The gall-bladder now has become a closed sac, the bile which it contained is gradually absorbed and replaced by a mucous or glairy fluid; this is at first so abundant as to convert the bladder into a tense capsule, resembling, according to Rokitansky, the sound of fishes, but, afterwards, this fluid is reabsorbed and the gall-bladder contracts and shrivels. Cholesterine is often present in great abundance in the fluid contents of such gall-bladders; this is especially the case when the coats are diseased and thickened. The loss of the biliary reservoir seems to have no injurious effect on the health. The duct. com. choled. may be closed by concretions, cancerous growths, or croupy exudation: the outflow of the bile being thus prevented, it collects within the ducts and causes general dilatation of them. In such cases Rokitansky says the liver is in a condition resembling that of yellow atrophy, the parenchyma of a dark yellow or green colour, turgid, though pulpy and friable. Dr. Budd, in the case he records, describes it as of a deep olive, finely mottled with yellow, the tissue flabby, but not easily broken down, the lobules undistinguishable. The cells in this and in another case were destroyed, and only granular and oily *débris*, mingled, in Dr. Budd's case, with yellow matter, remained. In two cases which we have observed, the cells were not destroyed; they had a yellow or greenish tint, and were rather stunted, but not at all broken up. We injected in one a large dilated duct, and obtained the important result *that the terminal ducts were not dilated, and were of about the same size as in healthy livers.* Rokitansky says that "this affection invariably proves fatal with symptoms of biliary infection of the blood and consequent cerebral disease, which is often combined with exudation on the arachnoid, with intense icterus, and extreme pain in the liver." In Dr. Budd's case there were no symptoms of cerebral poisoning, and the mind remained clear to the last.

Croupy inflammation occasionally, but very rarely, attacks the gall ducts. "It gives rise to tubular exudations, in which the bile forms branched concretions, which block up the passages, and thus cause dilatation of the capillary gall-ducts." The coats of the gall-bladder may become œdematous in dropsy, or the subserous tissue infiltrated, as in peritonitis; increased deposit of fat may also take place in the latter situation, and perhaps induce (coincide with?) fatty degeneration of the muscular layer. Rokitansky also notices the formation of osseous plates in the thickened parietes of gall-bladders which have been the seat of inflammation, and an increased production of fibroid tissue, which may be so firm and white as to give a cartilaginoid appearance. The biliary ducts very rarely contain tubercle. Cancer sometimes extends to the gall-bladder from the liver, or, it is said, may occur in it primarily; it forms nodules in the submucous tissue, or infiltrates the mucous membrane; more commonly its wall is perforated by growths in the liver, which push their way into its cavity.

BILIARY CALCULI.

Two conditions of the bile in the gall-bladder deserve our close attention, on account of the important consequences which often result from them. One is, the bile being so loaded with colouring matter from concentration, or other causes, that a deposit of this substance takes place; the other is, the bile containing a large quantity of cholesterine. From these two substances all biliary calculi almost are formed. They are far most common in the gall-bladder, but also occur in the ducts, both within and without the liver. "Their* form and surface vary much. Single calculi are commonly round, oval, or cylindrical; when very large so as to occupy" the entire cavity of the gall-bladder, they are frequently slightly curved; "if many exist together they mutually prevent their enlargement, and in consequence of the friction and pressure they exert upon one another they assume cubical, tetrahedric, prismatic, or irregularly polyhedric shapes, with convex or concave surfaces. The calculi found in the ducts are generally cylindrical, occasionally branched, or entirely amorphous. The texture of the calculi may be uniform or varied, in proportion as they consist of one substance or of several layers. Many show no distinct arrangement; some have an earthy pulverulent fracture, or a fibrous, striated, laminated, micaceous texture, as is particularly observed in calculi consisting of cholesterine." Gall-stones are not of any great degree of consistence, they may sometimes be compressed easily between the fingers: they are rather light, but not so much so as to float in water. Their colour varies from a milky white to various shades of green, yellow, or brown: internally they often present an alternation of different coloured laminæ. They may be said to consist generally of cholesterine, mingled with a combination of pigment and lime in various proportions. Large gall-stones,

FIG. 145.



Small irregular gall-stones, composed of inspissated and altered bile cemented by mucus. (From Dr. Budd's Work.)

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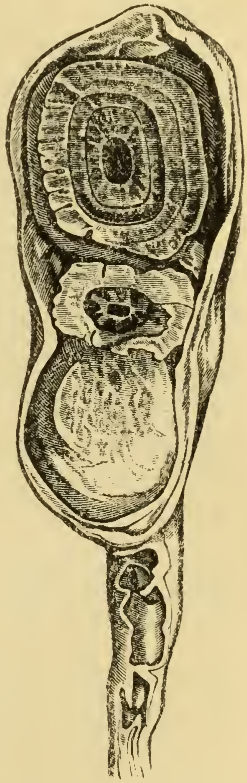
* Rokitansky, vol. ii. p. 162.

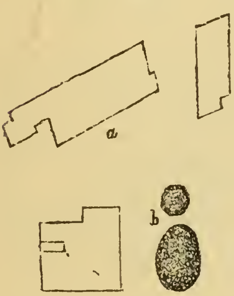
with the exception of their nuclei, consist almost entirely of cholesterine, and are therefore whitish and crystalline, their sectional surface presenting a number of striæ radiating from the centre. Small gall-stones, resembling grains of black pepper of an irregular tuberculated form and almost black colour, are occasionally found: they consist almost entirely of pigment and earthy matter, the carbonate and phosphate of lime. Cholesterine generally forms the principal mass of biliary calculi; it often alternates with layers of pigment, and almost always itself surrounds a nucleus of the same matter. Some are composed chiefly of carbonate of lime.

Fig. 147.



Gall bladder and cystic duct containing calculi, which have a crust of pure cholesterine. The two upper are divided. (From Dr. Budd's Work.)

Fig. 146.



From a gall bladder which was shrunken, a calculus being impacted in the cystic duct.
a, Cholesterine tablets, b, Glomeruli.

The secretion of a large quantity of cholesterine does not seem to be the essential and adequate circumstance for the formation of gall-stones: the gall-bladder sometimes contains mucus loaded with sparkling tablets of this substance, without any trace of calculi. It seems most probable that pigment granules cemented together by mucus first constitute a nucleus, round which cholesterine afterwards is deposited in layers. Other matters, however, may serve as a nucleus: blood, a portion of a distoma, or a lumbricus, or even a pin, are said to have been found in this situation.

Gall-stones are not peculiar to, or specially associated with, any condition of the liver—they are said to be most frequent with cancer, but very often occur in other states; they are more often found in females than males, in the proportion of 4 or 5 to 1,—rarely before, but often after the middle period of life. A sedentary life, and obese condition of body, are favourable to the formation of gall-stones; they are not, however, unfrequently present in lean and temperate persons. As cholesterine is a variety of fatty matter this might seem surprising, did we not remember that its formation may be referred to a kind of fatty degeneration, as well as to the presence of an increased quantity of oil in the system.

Cholesterine is certainly secreted often in large quantity by the thickened coats of the gall-bladder, and by additions from this source the large solitary calculi are probably formed; there is no doubt, however, that in other cases it is deposited from the bile, owing to the decomposition of the taurocholic acid or its salts, by which it is naturally held in solution. Calculi are often loose and free in the cavity of the gall-bladder; sometimes they are attached to its surface by exudation, or included in compartments formed by organized lymph. Small ones are sometimes, also, contained in saccular dilatations of the mucous membrane, and may appear to lie external to the cavity of the gall-bladder. The following effects are produced by biliary calculi. They become impacted in the cystic duct, and occasion its obliteration, the gall-bladder undergoing the changes that have been described. The same thing occurs, but much more rarely, in the common duct, which is straighter and wider; great distension of the gall-bladder and ducts, and occasionally rupture of the former, then take place. If on account of the angular shape of the stone the duct is only partially obstructed, the same effects are produced, but in a less degree. While lodged in the gall-bladder calculi may excite irritation, thickening, inflammation, and suppuration of its coats, and sometimes ulceration. Of these we have lately spoken.

Acephalo-cysts, and the distoma hepaticum, occur in the gall-bladder. For a description of them, see article Parasites, p. 214.

CHAPTER XXXVII

ABNORMAL CONDITIONS OF THE PANCREAS, AND THE OTHER SALIVARY GLANDS.

THESE are not very numerous. Congenital deficiency is observed only in very imperfect monstrosities, and excess of development is very rare. *Hypertrophic* enlargement, Rokitansky states, is altogether unusual, and when it does occur, affects chiefly the cellular tissue, which is interwoven with the glandular. We have, however, examined one specimen in which the ultimate vesicles were stuffed with epithelium to such a degree, that their investing fibroid envelopes appeared stretched and distended, and the whole gland was of a very remarkable density. We think minor degrees of this condition are not uncommon.

Atrophy of the pancreas takes place in some instances spontaneously, chiefly in advanced age; or it may result from chronic inflammation, or fatty degeneration. The organ may be soft and lax, or of leathery consistence.

Inflammation, at least in the acute form, but rarely attacks the pancreas; it is, however, not infrequent in the other salivary glands, where it constitutes the disease termed Mumps. We have seen inflammation and suppuration of the parotid gland occur as the result of fever. The phenomena of inflammation are the same here as in other similar parts. The gland swells considerably, partly from the congestion of its vessels with blood, partly from exudation into the areolar tissue which envelopes it. In the ordinary case of mumps suppuration rarely takes place, and simple resolution occurs; but when the result is less favourable, the glandular structure becomes, in a measure, fused with the interstitial exudation, and probably penetrated by it also, and the whole mass softens and breaks down into purulent matter. This had occurred in the case of fever above alluded to. The suppuration may affect the whole gland, or be limited to distinct spots, and form an abscess. Chronic inflammation of the parotid induces condensation, induration of the cellular tissue, obliteration of the acini, and either permanent enlargement, or subsequent atrophy of the gland. In such a case we shall find a very large quantity of coarse fibrous tissue surrounding and enveloping the lobes and lobules of the gland, the gland-tissue more or less atrophied, and the epithelium containing much oil.

A peculiar case of parotitis has been described by Dr. Wickham Legg, in which the gland was found pale and not at all hyperæmic; possibly through some change involving hypertrophy of the epithelium and anæmia, as in some cases of kidney disease. It occurred in the course of chronic morbus Brightii. ("Trans. Path. Soc.," vol. xx. p. 183.)

Fatty degeneration of the pancreas is described by Rokitansky as frequent in drunkards, associated with fatty liver. It is not, however, a degeneration of the same kind, but rather, from his account, seems to take place by intrusion of the surrounding adipose tissue on the wasting organ. A case of fatty degeneration has been reported as occurring in diabetes. True fatty degeneration was observed in a case where the gland was found lax and flabby with numerous dead whitish spots upon its surface. It was exceedingly degenerated, almost all trace of glandular structure being lost, and only an amorphous, granular, and oily mass remaining, in which were a great many fatty masses containing numerous fat-vesicles here and there. The body was exceedingly emaciated, all the fat in the abdomen and elsewhere being absorbed.* Serous cysts occur occasionally in the pancreas, and the other salivary glands.

Lardaceous degeneration may affect the pancreas, though rarely.

NEW GROWTHS OF THE PANCREAS AND SALIVARY GLANDS.

Cancer does not select these glands as one of its ordinary sites. It does, however, affect them not unfrequently both primarily and secondarily. Scirrhus and encephaloid are the only two forms which occur. It is probable that, in several of the cases reported as cancer of the parotid, the disease was really seated in some of the adjacent or imbedded absorbent glands. The head of the pancreas, where it is embraced by the duodenum, appears to be the part of the organ most frequently affected. As a result of the growth of the tumour, the ductus choled. is sometimes obstructed, and jaundice is produced. The disease may extend much further than this, according to Dr. Walshe, implicating "the duodenum, the omentum, mesentery, liver, and even the supra-renal capsules and kidneys." Rokitansky says, "that the secondary affections of the salivary glands, by an extension of the disease from adjoining organs, and in the case of the pancreas especially, by an extension from the scirrhous pylorus, is very common."

The pancreas is very little liable to new growth of any kind. Cancer is said to occur, though very rarely, as a primitive growth, and grows in the form of circumscribed tumours, while the rest of the gland becomes atrophied; though it may ultimately involve the whole. More often it spreads from some adjacent part, *i.e.*, from the duodenum, stomach, or lymphatic glands. No other form of tumour is known to originate in the pancreas. The retroperitoneal glands near the head of the pancreas and the surrounding connective tissue are occasionally the seat of a sarcomatous growth, which becoming closely united with the pancreas and compressing its ducts, has sometimes been described as a disease of that organ, and is functionally the same.

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* Handfield Jones, "Trans. Path. Soc.," vol. vi. p. 223.

The parotid gland is much more commonly the seat of new growth. One form of tumour very characteristic of this part is *myxochondroma*, a soft form of cartilaginous tumour, in which the cartilage is intermingled with soft mucous tissue like that of a myxoma, and sometimes with cellular masses which would be called sarcoma. Förster states that it is sometimes combined with cancer, and mentions a case in which such a tumour was truly malignant, and produced secondary growths in distant parts. We might naturally suppose that these tumours do not arise in the gland itself, but in an adjacent lymphatic or in the connective tissue; nevertheless, secreting gland-tissue is sometimes found imbedded in the tumour.*

We have also seen a spindle-celled sarcoma; and an internal nævus or angioma has been described from the same situation.† True glandular tumours or outgrowths of parotid tissue not unfrequently occur.

Cancer may commence in the gland itself, in the surrounding connective tissue, or in one of the neighbouring lymphatic glands. It forms sometimes very large masses, which may grow downwards to the clavicles, inwards to the vertebral column, or behind the œsophagus, and sometimes ulcerate through the skin. Scirrhus and encephaloid occur most frequently, but epithelioma has also been described. Cancer of the submaxillary or sublingual glands appears always to be an extension from the neighbouring lymphatics.

Salivary fistulæ are usually caused by the progress of ulceration. Thus, a perforating ulcer of the stomach may make its way into the pancreatic duct, and a like event may occur to the duct of Steno, which is oftenest perforated by extension of ulceration from the mouth.

Dilatations of the ducts are produced in consequence of obstruction of their outlets, while the secretion accumulates and distends the canal. The obstruction may depend on an external tumour, or a mucous plug, or on a calcareous concretion. Sometimes the dilatation occurs at several separate points; sometimes it forms fusiform "or closely-set expansions, partially separated from one another by valvular folds formed by the coats of the duct." Dilatation of the duct of the submaxillary gland, or Wharton's duct, produces a tumour within the mouth which has received the name of *Ranula*. The salivary concretions, or calculi, are described by Rokitansky as "white, friable, and either round, oblong, cylindrical, or obovoid; in size varying from that of a millet seed or pea. to even that of a hazel nut. They are either solitary, or, if small, frequently very numerous (twenty and more); and they are composed of phosphate and carbonate of lime, held together by animal matter." The saliva from which they are formed by deposition, must be, as Dr. Walshe remarks, in an unhealthy state; for while the concretions consist chiefly of phosphate of lime, sometimes containing 94 per cent., there exists very little of this salt in the normal secretion. "It becomes, therefore, extremely probable that the excess of phosphate is generated through the influence of irritation of mucous membrane."

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* For cases with figures see Arnott, "Trans. Path. Soc.," vol. xx. p. 186; and S. Jones, Ibid., vol. xxiii. p. 263.

† Gascoyen, "Trans. Path. Soc.," vol. xi. p. 267.

ABNORMAL CONDITIONS OF THE SPLEEN.

This organ is generally absent in acephalous monsters; sometimes it is wanting, together with the stomach, or the fundus of the stomach, in subjects otherwise well developed; or it may be itself alone in a rudimentary state. Small supernumerary spleens, which are often met with in the vicinity of the organ, are not to be regarded as instances of its multiplication, but of its subdivision. The spleen is liable to very great variations in size, probably more than any other organ of the body. This depends chiefly on the very large size of its vascular system, and on the great quantity of yellow, elastic fibre contained in its structure, which allows it to be distended to a prodigious extent. Most, if not all, hypertrophies of the spleen, however, are produced not only by engorgement of the vessels, but by an alteration and increase of the red, pulpy parenchyma which they traverse. This parenchyma, consisting chiefly of lymphoid corpuscles, has certainly very close relations with the vascular system of the organ, some histologists asserting that the blood finds its way through spaces which have no regular walls. Whether this be the case or not, it is certain it will very readily admit of increase or diminution corresponding with the distension of the vessels. Its corpuscular elements appear to be also susceptible of rapid increase.

We shall here only mention the degrees of change in size which the spleen may undergo. Rokitansky states, that "the spleen not unfrequently measures sixteen inches in its long, seven inches in its short, diameter, and four inches in thickness; its weight may amount to 13¼ lbs.," or even, as others affirm, to 20 lbs. and upwards. One weighing 13 lbs. is reported by Dr. Squire ("Trans. Path. Soc.," vol. xxii. p. 276), from a case where the white corpuscles of the blood were increased; another, described by Mr. Nunn (Ibid., vol. xiv. p. 252), weighed 13½ lbs.; and was not associated with any marked symptoms. The opposite change of atrophy may reduce the spleen to the size of a hen's egg, or a walnut. The *form* of the spleen is rather various. It may be tongue or platter-shaped, or cylindrical or globular. One of the most important circumstances to note under this head, is the great frequency of notches in its anterior border, which may be felt through the abdominal parietes when the organ is enlarged. The spleen is liable to various *displacements*, some of which are congenital, others the result of disease. Haller found it lying by the side of the bladder in a child one year old, Desault in the right side of the thorax in a new-born infant. It has been found in the left thoracic cavity when the diaphragm was absent, and external to the abdomen in large umbilical herniæ, or where the abdominal parietes were fissured. Displacement may ensue from the enlargement or distension of adjacent parts, or from its own increase in size. Sometimes in the latter case it descends to, and slides off, the ilium, "so as to occupy a diagonal position in the hypogastrium, and extend over the right ilium."

Hypertrophy of the Spleen.—Enlargement, without any notable change except hyperæmia, takes place in many continued fevers and acute constitutional diseases, as enteric fever, erysipelas, pyæmia, puerperal fever, &c.

It then presents the following characters. The enlargement may be to twice or four times the normal size, the capsule appears tense, the gland plump and elastic, but on cutting it open it is soft and sometimes almost liquid, very vascular, and of a dark-red colour. If firm enough to form a distinct section it is either uniformly smooth, or more often coarsely granular. Microscopic examination (according to Förster) shows abundant new formation of small lymph cells and nuclei, to which, rather than to the vascular fulness, the increase of size is due. The newly-formed cells and nuclei are often contained in large compound mother cells, which are found in various hyperplastic conditions in the spleen pulp and in the splenic vein.

In other cases we have found, under the microscope, no very noticeable alteration of the structural elements. There may be some increase in the quantity of diffused granulous matters; but the nuclei appear quite natural. The change is one better judged of on a large than on a small scale. We have examined, at different times, numerous specimens of greatly enlarged spleen, but we have found little that could be regarded as characteristic of the several alterations. This is not surprising; it could not be expected that specific differences in the blood should mark themselves by corresponding varieties of form in the cell-growths of their exudations, any more than that the syphilitic virus should be detected by some special modification of the pus of a chancre.

Chronic hypertrophy is seldom unaccompanied by some other change; but cases of apparently simple hypertrophy do occur, such as was the spleen weighing 13½ lbs., before-mentioned, where nothing abnormal was found in the spleen or in the blood.

In *Rickets* the spleen is often enlarged, when the disease has lasted a long time. The enlargement appears to be accompanied by something resembling albuminous infiltration, distinct from lardaceous disease.

In *Syphilis*, especially in the congenital form, there is sometimes enlargement, with or without distinct tumours. In *scrofulous* disease, and *tuberculosis*, a general enlargement is also spoken of.

Obstructive disease of the heart sometimes produces enlargement with hyperæmia of the spleen; but the engorgement is less marked than in the case of the kidneys, liver, and other organs, whose veins open into the vena cava direct, since, in the case of the spleen, the liver intervenes to break the force of the backward pressure.

In *intermittent fever* enlargement of the spleen is constantly observed. The hypertrophy is accompanied by abundant deposit of pigment, orange, brown, or black, in free granules through the pulp, or sometimes enclosed in cells. It may also be deposited in the Malpighian bodies, and sometimes thus produces a remarkable spotted appearance. The frequent occurrence of pigment in the spleen is probably due to its liability to hæmorrhage, which is in all organs so constant a cause of pigmentation.* Acute swelling of the spleen is said also to accompany the febrile paroxysm, or ague fit.

In *Leucocythemia* constant enlargement of the spleen takes place. The substance is often quite natural, sometimes paler than usual, and sometimes with a peculiar smooth lustrous appearance. There appears to be a general hypertrophy of the organ in all its parts. The largest spleens known are found in connection with this disease. Separate masses of lymphatic structure sometimes occur in the spleen in this disease. They are white, crumbling or soft, and composed chiefly of lymphatic corpuscles.

Great enlargement of the spleen is seen in the disease before referred to as *Hodgkin's disease*, or *lymphatic anæmia*, in which the lymphatic glands, and often the liver, are also enlarged. The spleen is found large and hard; while contained in its substance, and projecting on the surface, are white or yellowish masses, varying in size, of firm, tough, glistening substance, rarely soft and opaque, not crumbling. The larger are often irregularly conical, like infarctions. Beside these masses ordinary infarctions, with obstructed arteries, are sometimes found. The adventitious masses consist of lymphoid cells imbedded in a stroma, like that of a lymphatic gland, which sometimes becomes amorphous, granular, or translucent, as if infiltrated with waxy material, but is never stained by iodine.*

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* We may here repair an omission made above (p. 492) in speaking of pigment in the lungs, where it is made to appear that all the pigment in the lungs is carbon introduced by respiration; whereas a large portion is doubtless due to congestion and hæmorrhage, as already stated on p. 163.

Lardaceous degeneration of the spleen frequently occurs simultaneously with the same disease in other viscera. It usually commences in the Malpighian corpuscles, which become enlarged into translucent granulations an eighth of an inch or more in diameter, much resembling grains of sago, whence the spleen in this condition has been called the "sago spleen." In later stages the whole structure becomes infiltrated with translucent material, and in both cases the substance is firm, tough, and anæmic. There is a constant, and sometimes very considerable, increase of size. Iodine produces the characteristic reddish-brown colour.

Atrophy of the spleen most commonly occurs as a senile change, but is also found independently of old age in various wasting diseases, especially those accompanied by anæmia, unless there happen to be any disease of the organ. The size of the organ is also reduced, probably by pressure, in abundant peritoneal exudation; and in chronic peritonitis, unless there be any other condition tending to enlarge it. It would seem as if even acute peritonitis had some tendency to prevent enlargement of the spleen, which might otherwise occur in some febrile diseases. Atrophic spleens differ much in colour, vascularity, and consistence, but are frequently more fibrous than natural.

Hyperœmia of the spleen occurs both from mechanical causes, and from the causes enumerated as producing acute enlargement. Rokitansky remarks that though it occurs in organic diseases of the heart and in hepatic obstructions, it does not amount to the extent, nor take place so frequently as might be expected, and he accounts for this by the deranged circulating fluid having no affinity for the tissue of the spleen. Distension of the abdomen with fluid appears to have some effect in preventing the swelling and hyperæmia which would otherwise occur. In the bodies of drowned persons the spleen is found gorged and distended with blood. This blood, no doubt, might all be washed out by injecting the vessels with water, and the organ would return to its normal size; but when congestion comes to be permanent, the exudation which takes place in the red parenchyma causes multiplication of the normal cells and nuclei, and the spleen is then truly hypertrophied.

An *anœmic* state of the spleen is observed in the highest degree when the parenchyma is infiltrated with lardaceous matter, before noticed; but it also exists in many atrophic conditions, as in old age.

Wounds and Ruptures of the spleen occasionally happen from injuries or accidents. The only point of interest respecting these is, that there appears, from the observations of Mr. Athol Johnson, ground to believe that under careful management they do not necessarily prove fatal. Spontaneous ruptures of the spleen have also occurred in conditions of intense congestion, and when the texture of the organ was probably weakened, as in typhus, cholera, and the cold stage of ague. These always prove fatal. Such accidents suggest the prudence of handling the abdomens of fever patients cautiously.

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* Cases may be found reported in "Trans. Path. Soc.," vol. iv. p. 177; vol. x. p. 259; vol. xi. p. 257; vol. xi. pp. 247, 269; vol. xix. p. 401; xxii. p. 278. Also in "Guy's Hospital Reports," Third Series, vol. ii., by Dr. Wilks.

Inflammation.—" Primary inflammation of the spleen," Rokitansky says, " is as rare as spontaneous primary phlebitis; secondary, as frequent as secondary phlebitis." Primary inflammation of the spleen, unless ending in resolution, gives rise to the production of laudable pus or fibrine. The pus may be contained in a circumscribed abscess, and thence become obsolete, or the cavity may go on enlarging until the abscess makes its way into the left thoracic cavity, the stomach, the transverse colon, or the peritoneum. When the latter event happens, circumscribed peritonitis often forms a sac for the pus with the aid of the surrounding parts. The causes and pathology of spontaneous splenitis are very obscure. The secondary splenitis of Rokitansky seems to be identical with pyæmic abscess. The deposits are well defined, always situated at the periphery, usually of a cuneiform shape, the apex directed inwards; their colour is considerably darker than the surrounding tissue, and their consistence firmer. A ring of reactive inflammation is often set up around them. The process may (according to Rokitansky and Förster) terminate either in the case of a benignant fibrinous exudation in conversion of this into a cellulo-fibrous callus, which contracts and causes a cicatrix on the surface; or in the case of a less healthy exudation in the conversion of this " into a puriform, creamy mass, or into a sanious, greenish, greenish-brown, or chocolate-coloured pulp."

The pyæmic blocks have much resemblance to the simple or fibrinous blocks, and have doubtless often been confounded with them, while the actual healing of pyæmic abscesses, though described by good authorities, seems open to doubt. True pyæmic blocks, or secondary abscesses, seem to differ from the simple blocks in their rapid suppuration, irregular shape, and in the constant inflammation of the capsule when they are near the surface.

Hæmorrhagic Infarctions or Fibrinous Blocks.—The blocks or infarctions of the spleen are among the most important and characteristic morbid appearances, being not found so often elsewhere, except in the lungs and the kidneys. As explained before, their occurrence depends on a peculiarity in the anatomical arrangements of these organs. In each of them are terminal arteries, which form no further anastomosis one with another, so that if one be blocked up the circulation cannot be restored, and they have veins without valves, which permit regurgitation. An infarction is essentially a part of the organ where the circulation is suspended, and where those changes are produced which were before shown to ensue on this obstruction. In the spleen it will be found that the blocks correspond to the areas marked by the terminal division of branches of the splenic artery, where they break up into the diverging brush of small twigs called the penicillium. The blocks are accordingly wedge-shaped or pyramidal, with the apex towards the hilus of the spleen; and in the typical cases coinciding with the ultimate breaking up of the artery. Coagulation once set up in this space may, however, extend beyond its limits, so that the shape becomes irregular. The cause of the arrest of the circulation must be, in most cases, the obstruction of the artery, but it is not quite clear whether a somewhat similar condition may not result from blocking of the vein.

The arterial obstruction is not always produced by the same cause. Sometimes it is due to embolism, and hence, in a large number of cases of blocks in the spleen, we find cardiac disease, and especially vegetations on the valves. But sometimes no such antecedents can be demonstrated, and it appears that there must be spontaneous coagulation of blood in the artery, perhaps assisted by some diseased condition of the arterial wall, or from some morbid condition of the blood. This is especially the case in fevers, where blocks sometimes occur without any source of embolisms. In either case infarction results in the manner already traced, and the block, once formed, goes through the changes described above (p. 397).

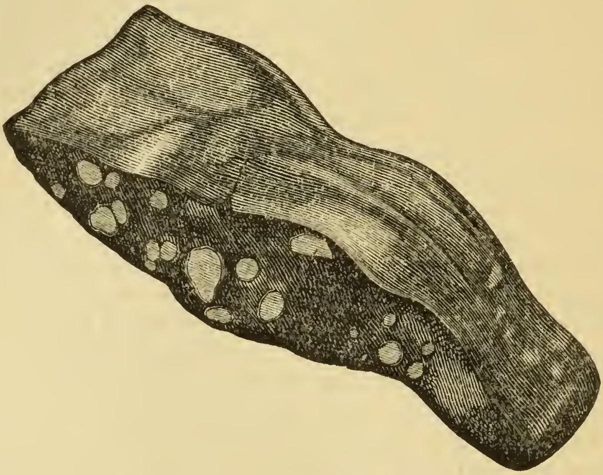
In the end, a mass of fibrous tissue results, which shrinks into a cicatrix, and after a time nothing but a depression on the surface may remain, unless, as sometimes happens, calcification occur. The accompanying hyperæmia and inflammation always cause some swelling of the gland, which, if the blocks are numerous, as they often are, may be very considerable. This is, perhaps, the most frequent cause of the splenic enlargement in cardiac disease.

Cancer and New Growths.—Primary cancer is so exceedingly rare that it may be doubted whether any unquestionable case has yet been reported. Secondary cancer occurs chiefly as the sequel of cancer of the stomach, or some other abdominal viscus; but even this is very uncommon.

Of simple tumours, lymphomata or lymphatic growths, already spoken of, are the commonest, and appear sometimes to have their primary seat in the spleen. Fibroma and angioma, or cavernous tumour, have also been described. Isolated masses of splenic tissue, forming distinct tumours, are sometimes observed.

Tubercle occurs, for the most part, in the spleen only in acute universal tuberculosis; it occurs more frequently in children than in adults, in the proportion of 40 to 13. It appears both in the form of grey granulations, yellow miliary tubercles, or yellowish cheesy masses, of the size of a pea and above. In acute tuberculosis the spleen is described by Rokitansky as becoming swollen and softened, much as in a typhous state. The tubercles are here usually hard, grey, and like those in other organs. But in cases which are rather more chronic, the spleen often contains tubercular masses larger than those in other organs; and it seems as if a single tubercle were capable of attaining a larger size in the spleen than elsewhere except in the brain.

FIG. 148.

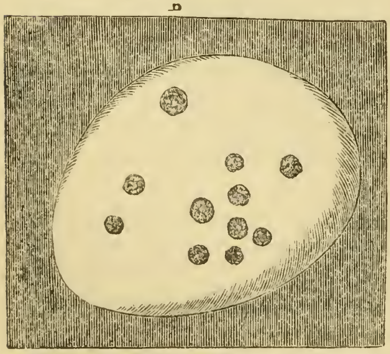


Masses of yellow tubercle in spleen.

Syphilitic gummata are occasionally seen in the spleen.

Cysts.—We have occasionally observed small cysts in the spleen, or, perhaps, to speak more accurately, in its capsule; in a female of middle age dying with fever, there were several small, firm nodular prominences on the anterior border; they were of conical shape and lightish red colour, appearing like so many growths on the surface; under the microscope they were found to be small cyst-like cavities of varying size (one measured $\frac{1}{66}$-inch diameter), oval or spherical, containing numerous large granule-cells floating in a transparent liquid.

FIG. 149.



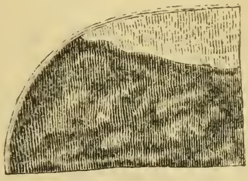
Cyst in the capsule of the spleen, containing a clear fluid and glomeruli.

Hydatid cysts are sometimes found in the spleen alone, or, concurrently with one in the liver. It rarely attains the size it reaches in the latter organ. Dr. Coley exhibited to the Pathological Society a specimen of the magnitude of a cocoanut. Pentastomum denticulatum was found in one case incapsulated and calcified by Wagner.

Dermoid cysts, containing fat and hair, have in rare cases been observed.

Changes in the Capsule.—The fibrous capsule of the spleen is very frequently the seat of *chronic fibroid thickening*, which sometimes proceeds to a very great extent. The thickened membrane has much the aspect of cartilage, but none of its real characters. Usually the thickening extends pretty uniformly over the surface, leaving, however, here and there, spots less affected; sometimes it forms nodular masses grouped together, and sometimes patches which look like drops of tallow deposited on the surface. In one case we examined carefully, it was very apparent that the thickening had taken place on the inner surface of the capsule, at the expense of the parenchyma, and we are inclined to think this is generally the case. Certainly the process is distinct from the formation of bands of adhesion to adjacent parts. A thickening of the capsule may be all that remains of a wasted block. Ossification of the thickened fibroid layers takes place, though rarely, and chiefly in very old persons.

FIG. 150.



Fibroid thickening of capsule, encroaching on the dark parenchyma.

Vertical section.

Morbid Conditions of the Vessels.—Extreme atheroma of the splenic artery leads as in other cases to wasting of the organ. Thrombosis of the vein occurs not unfrequently in leuchæmia; and sometimes in other conditions, especially, as it is said, in suppuration. Phlebolithes may form in the venous channels thus obstructed.

ABNORMAL CONDITIONS OF THE THYROID GLAND.

We are not acquainted with any instance of excessive congenital development of this gland, unless the rare occurrence of simple hypertrophy should be regarded as such. Mr. Curling has recorded in the "Medico-Chirurgical Transactions" (vol. xxxiii.), two instances of its absence in idiots. The most complete account of its morbid alterations is that given by Professor Hasse, from which we shall extract the greater part of the following summary: —"*Inflammation* of the thyroid is rare. It may attack the organ either when healthy or when enlarged by previous disease. Its course is more frequently chronic than acute. Within a very brief interval the gland often swells considerably,—becomes very blood-shot, tense, and painful,—its texture softened and friable, assuming first a brown-red, and ultimately a dingy-grey colour." When suppuration occurs, there may be several foci, or one large one involving the whole gland. The abscess may open externally, or into the œsophagus, or into the trachea, as in a case figured by Dr. Baillie. After this has occurred the gland on the side affected shrivels "into a hard, cellular, filamentous knot, which adheres firmly to the skin and the surrounding parts. Sometimes the shrivelling of the one gradually brings on wasting of the other lobe." Andral states that acute tumefaction of the thyroid may come on after violent exertion.

Simple enlargement of the thyroid is frequent, says Hasse, and for the most part inconsiderable; but it implicates the entire gland, and thus may cause greater disturbance than a more extensive tumour of another kind. "Both lobes of the gland, and even the middle one, swell so as to encroach equally on each side, against the trachea and vessels of the neck." The affection is almost wholly confined to youth, and is frequent about the age of puberty in both sexes—more so, however, in the female, in whom enlargement is especially apt to prevail at the approach of the menstrual period. The essence of simple hypertrophy seems to be the derangement of the equilibrium naturally existing between the processes of secretion and absorption that continually go on in the gland. When the former predominates, the vesicles, and of course the whole organ, become distended.

Enlargement of the thyroid, especially of its vascular system, accompanied by violent pulsation of the heart and prominence of the eyeballs, constitutes the disease known as exophthalmic goitre.

Chronic Enlargement, Bronchocele or Goitre.—In the chronic and permanent enlargements of the thyroid a more considerable amount of change takes place. The vesicles of the gland are not only distended by excess of their natural secretion, but, besides being greatly enlarged, are filled with calcareous, atheromatous, and other matters. It is probable that some new vesicles are formed as well as old ones enlarged. Hasse makes two separate varieties of melicerous degeneration and cystic formation; but we think they run so much into one another that they may be classed together. In the pure melicerous degeneration the secretion which distends the vesicles, and some of them much more than others, is a tenacious, jelly-like substance, of the colour of honey. The vessels seem to be usually compressed, so that the gland appears bloodless. The change is sometimes limited to certain portions, an interesting instance of the power which the vitality of each individual elementary part has in determining the character of its actions. In the more common cases of enlarged thyroid the vesicles contain, beside colloid matter, more or less inspissated or fluid, varying quantities of calcareous, often ossiform substance. Tablets of cholesterine and other fatty matter seem to be not unfrequently present, probably as the result of fatty degeneration. An instance related by Dr. Häen shows the great variety of appearance which the contents of the cysts may present. He says that in a frightfully enlarged thyroid he found almost every variety of tumour existing together. "Here was a steatoma, there an atheroma, in another place a purulent tumour, in another an hydatid; in one there was coagulated, in another fluid blood, on this side was a loculus full of glutinous matter, on that was one filled with calcareous matter mingled with tallow." One of the cystic cavities sometimes enlarges prodigiously at the expense of the others; Andral relates having found a thyroid transformed into a cyst with bony walls filled with a honey-like matter.

Another kind of enlargement of the thyroid consists in the *dilatation of its vessels*, constituting what is called vascular or aneurismatic bronchocele; though, according to Förster, this term has been applied to ordinary bronchocele when very vascular. The veins in particular, writes Hasse, form very dense, capacious, often knotted plexuses, and the whole texture consists, apparently, of a dense coil of vessels. The substance of the gland has almost entirely lost its granular character—it is flabby and dark-red. After death, the tumour collapses considerably. The walls of the arteries and veins are attenuated; the dilated membranes of the vessels contain considerable clots, and capacious cavities are found filled with black coagulated blood. In a remarkable specimen which seemed in part to belong to the above class, we found the greatly dilated vessels completely coated with oily matter, so as in some parts to appear like white cylinders by direct light. At the same time the glandular vesicles were destroyed, and only some traces of their epithelium could be discovered; a most important feature of the alteration, which is not mentioned by Hasse in the above description, though it seems inferable, from his statement, that the granular (vesicular) character of the gland is almost entirely lost.

New Growths.—Tubercle is very rarely found in the thyroid; a case is, however, figured by MM. Cornil and Ranvier.

Cancer is also very uncommon, but when it does occur is more often primary than secondary. The growth usually affects part only of the organ, and owing to its situation may cause death by compression of the trachea, or may ulcerate into the air passages or œsophagus. Probably for this reason the disease has, in the few cases recorded, generally remained confined to this organ, and not become distributed over the body. In a case observed by the editor, medullary cancer arose somewhat rapidly in a thyroid which had for many years been affected with ordinary bronchocele; and it appears, from instances quoted by Virchow, that this has been the case in several of the few recorded cases of thyroid cancer.*

ABNORMAL CONDITIONS OF THE THYMUS.

Absence of the thymus in infants has only been observed in cases of acephalism. Inflammation is an exceedingly rare occurrence; but Hasse refers to two cases, in one of which an abscess is said to have opened into the trachea. In cases of tuberculosis the thymus is occasionally involved; it becomes considerably enlarged, "firmly united with surrounding parts, and either converted by tubercular infiltration into a hardened mass, or else partially destroyed by softening." Mr. Simon mentions a case in which suffocation was occasioned by the pressure of a tumour apparently of sarcomato-cystic character in the situation of the thymus, and Sir Astley Cooper met with a case of encephaloid growth in this part.

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* "Trans. Path. Soc.," vol. xxii. p. 283. Virchow, "Krankh. Geschwülste," vol. iii. p. 50.

The thymus is not unfrequently found greatly enlarged; but the nature of the hypertrophy, as it is called, has not been exactly determined by microscopic observation. It is tolerably certain that this enlargement is not, as has been supposed, the cause of attacks of sudden and sometimes fatal dyspnœa occurring in children. For it has been shown that "thymic asthma," as it is termed, may occur with an unnaturally small thymus; and that the gland may be greatly increased in size without producing any symptoms of dyspnœa.

The thymus is the starting-point of some of the lymphatic mediastinal tumours before referred to. They appear to begin with a simply homologous growth, since the structure of some of these growths resembles that of the normal thymus. Generally they remain simple tumours, but in some cases infiltration of neighbouring parts and of the lungs occurs, and the growth is decidedly malignant. The cancers formerly described as cancers of the thymus were probably of this kind.

ABNORMAL CONDITIONS OF THE SUPRA-RENAL CAPSULES.

The supra-renal capsules may be deficient, especially where there is a deficiency in other organs also. Their absence, however, does not coincide with that of any other organ in particular, and they may be present when one kidney is absent, so that their name must not be taken to imply any correlation of function. Accessory supra-renal capsules are of frequent occurrence. They are sometimes found *hypertrophied*, but the nature of the enlargement does not appear. It is said that they are unusually large in negroes, and that the cortical substance contains an unusual quantity of pigment. Their normal condition is one of at least relative *atrophy*, that is, they do not grow and increase in size together with the other parts of the body.

The principal indications of atrophy are manifested, we think, by the shrinking and diminution of the medullary substance, and the breaking up of the cortical cells into oily masses. It is interesting to compare the condition of the degenerating capsule with that of an hepatic lobule. In one instance, where a careful search was made, at our request, for the supra-renal capsules in the body of a child six years old, who had died from a burn, no trace of them could be found, but a small quantity of lax, dirty-looking, reddish, infiltrated, areolar tissue, which presented under the microscope only a mixture of altered granulous nuclei, large granulous or oil-holding cells, and a very large quantity of diffused granulous matter imbedding some oil drops. This was a case of unusually early atrophy. Another indication of atrophy is afforded by the apparent formation of a central cavity, which appears to exist in some cases quite distinct from that of the vein. It is formed, we believe, by the wasting of the medullary substance, and the production thereby of a space which is traversed by blood-vessels, and occupied only by serum, and a little stromal fibre. In this way a lax spongy tissue is formed, which softens by *post-mortem* decomposition, or is easily torn, and the appearance of a cavity thus produced.

Hœmorrhage.—Rokitansky says, "hæmorrhage not unfrequently occurs in them, on account of the vascularity of their medullary substance. The capsule is found distended in proportion to the amount of extravasation caused by the rupture of a vein; and according to the period which has elapsed since the occurrence of hæmorrhage, we find the blood more or less discoloured and changed in constitution, inclosed within the cortical substance, which has become pale and atrophied, and is finally converted into a fibroid layer." *Suppuration* and *induration* are occasionally met with as results of inflammation of these glands. They have been found converted into purulent pouches in the newborn child and in the fœtus.

Constant Alterations.—The changes which occur so constantly as almost to be regarded as normal, are fatty degeneration and pigmentation. The former affects the cortical portion, where fat is found deposited in the cells. Though regularly present in the adult, this fat is said by Klebs to disappear from the cells in old persons, and also in the indurative conditions dependent on venous congestion or other causes. It is difficult to recognize any *excess* of fatty change as pathological. Pigmentation is also found in the cortical portion, but affects especially the inner layers of this, nearest the medullary portion. The pigment is deposited in a granular form in the cells; it is, in masses, brown, but in small quantity appears yellow. The pigmented portion becomes broader in old age; and with the increase of pigment becomes softer, so that it is easily broken down on removal from the body, and appears as a brownish detritus. This pigmentation has been supposed to be connected with the excessive coloration of the skin observed in some diseases of these organs; but if there is any relation, it must be one of antithesis, since when the skin is thus darkened, there is usually absence of pigment in the capsules.

Lardaceous degeneration often affects the supra-renal capsules when present in other abdominal viscera. The organ is enlarged, pale, hard, the medullary portion generally translucent, the cortex more often opaque and fatty. In less advanced cases the central portion only may be affected. Iodine produces the usual coloration, affecting chiefly the blood-vessels and partly the fibrous septa. No special symptoms are known to be connected with this condition.

Tumours and New Growths.—Cancer occurs both primarily and secondarily. The primary occurrence is not common. Klebs describes a case in a child aged eleven, where both the capsules were converted into tumours, one the size of a fist, the other smaller, of typical cancer, with angular cells contained in alveoli. Numerous secondary tumours had been formed in the mesentery, liver, and lungs. Another primary cancer of one supra-renal is described by Dr. Ogle, from a child ("Trans. Path. Soc.," vol. xvi. p. 250). Secondary cancers are more often observed subsequent to the same disease in the stomach, kidney, mamma, &c., and may affect both or one only. Sarcoma has rarely been described as such, but doubtless some of the cases of so-called cancer belong here ("Trans. Path. Soc.," vol. xviii. p. 260). It is important to observe that these organs are one of the few original seats of melanotic sarcoma. Cavernous tumour or angioma has been observed in one case, and a cavernous lymphangioma in another. No characteristic symptoms are known to be connected with any of the above changes, or only a slight discoloration of the skin different from that seen in the true Addison's disease.* The cases collected by Dr. Addison, in his original paper, included two of cancer; but subsequent observation has made it evident that these should be separated from the typical cases of the disease which is associated with his name.

Syphilitic growth has been observed in one case of congenital syphilis.

Tubercle occurs, though not very often, in the miliary form in cases of acute general tuberculosis. In chronic cases, masses of yellow cheesy matter are sometimes found, constituting what was formerly called scrofulous matter or yellow tubercle. This has much resemblance to the morbid changes in Addison's disease; but we wish to draw attention to the fact that such material may be found in the one, or even in both, supra-renals, independently of that disease ("Trans. Path. Soc.," vol. xxi. p. 200), though not without similar morbid products in other organs. Virchow has given the best description of tubercle in these organs. He describes it as beginning in the medullary portion as miliary tubercles, which become confluent and soften down; while the process extends to the remainder of the organ. He regards this as the beginning of Addison's disease, but the case on which he builds this hypothesis seems to have been one of acute tuberculosis, and did not present unquestionable symptoms of Addison's disease; this question is again discussed below.

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* See twenty-two cases collected by Dr. Greenhow ("Trans. Path. Soc.," vol. xvii. p. 316) of undoubted cancer, where this assertion is strictly borne out.

Cysts.—A cystic condition of these organs is described by Virchow as struma supra-renale; and a form of lymphatic cavernous tumour (lymphangioma) has also been described; but both are very rare.

Addison's Disease; or Melasma Supra-renale.—This name is given to the disease first distinguished by the late Dr. Addison, in which a peculiar degeneration of the supra-renal capsules is associated with extreme cachexia and peculiar pigmentation or bronzing of the skin. These three characters are nearly constant, but of the two latter, one, more often the cutaneous affection, is sometimes wanting, though the anatomical characters of the supra-renal bodies and the general course of the disease may be enough to identify it. We are here only concerned with the supra-renal bodies, the changes in which, though not always quite the same, are constant in certain features. The organ is always enlarged, its fibrous envelope thickened and adherent to the surrounding parts. The substance, hard, nodulated; all distinction between the cortical and medullary portions lost; and the natural structure quite obliterated. The adventitious material which occupies the place of the normal structures appears in two forms at least. *First.*—A translucent fibroid material, sometimes quite resembling cartilage in appearance, of a greyish or sometimes slightly greenish colour, said by Dr. Greenhow to "assume a pinkish hue on exposure to the air." *Secondly.*—White or yellowish opaque crumbling material, resembling "scrofulous matter" or "crude tubercle." Beside these two substances are seen collections of creamy liquid, like softened tubercle, and sometimes cretaceous matter; the two latter being apparently derived from metamorphosis of the whitish crumbling material. The different matters are sometimes mingled together so as to produce a mottled appearance, one passing imperceptibly into the other, sometimes the translucent material forming a diffuse infiltration, and especially becoming continuous with the thickened envelope, while the white opaque matter is enclosed by it in distinct masses. The general naked-eye appearance seems to us more like that of certain syphilitic gummata, especially of the brain, than any other morbid product. In typical cases of Addison's disease, when the change is far advanced, certainly no miliary tubercles are ever seen.

Minute Changes.—Microscopical examination of these products has yielded rather unsatisfactory results. The grey fibroid material is found to consist of connective tissue, with a granular fibrillated matrix, nuclei, and sometimes spindle-cells; but is sometimes nearly amorphous, perhaps from secondary degeneration. This appearance has suggested the term lardaceous to some observers, but it is quite distinct from lardaceous disease properly so called, and is unstained by iodine. The opaque portions consist of material obviously in the state of necrosis or decay: shrunk and withered cells, like what were formerly called "tubercle-corpuscles," amorphous granular matter, and fat molecules without any organizing structure to connect them. The creamy puriform liquid is not true pus, but débris of tissue, granular matter, and fat, like softened caseous material from other sources. Occasionally residual portions of the original structure, especially of the cortical cell-cylinders, are found imbedded in the new growth. Sometimes a small-celled infiltration is seen between these degenerated masses. This has been compared to interstitial hepatitis (Burdon Sanderson).

Relations to Tubercle.—The most important anatomical question with respect to these changes is, whether or not they are due to the original production of tubercle. Although miliary tubercle is rarely found in the organ itself, and perhaps never in advanced cases, it frequently occurs simultaneously in other organs, and it is not to be denied that the appearances closely resemble those seen in old pulmonary phthisis, or other tubercular disease, where the fibrous and caseous metamorphoses go hand in hand; so that nothing disproves the possibility of the disease having been tubercular in the first place. Again, without calling it tubercular, we might class it with the degenerative inflammatory conditions, called scrofulous, like some forms of pulmonary phthisis. Finally some pathologists regard it as an affection *sui generis*—neither scrofulous nor tubercular.

It has been described as tubercular by the majority of English observers, as well as by Virchow, Rokitansky, and Rindfleisch. On the other hand, Wilks, Burdon Sanderson, and others describe it as different from tubercle. Greenhow thinks there must be some intimate relation between this lesion and the tubercular diathesis. Klebs regards the question as uncertain, and describes it by the neutral name of fibro-caseous change ; believing some cases to be tubercular, others decidedly not. The fact seems to be, that in well-marked cases tubercles proper are never found, but that the appearances are not inconsistent with what would be found in late stages of the diseases commonly associated with tubercles. Out of 128 unquestionable cases tabulated by Dr. Greenhow, forty-six were virtually uncomplicated, seventy-two were complicated with some tubercular or scrofulous disease, while ten were combined with some other serious disease, but not with tubercle; so that in 56 per cent. tubercle was present in other organs.*

Nature of Addison's Disease.—With regard to this question, which is far too complex to be fully discussed here, we can only say, that it seems to be now proved that the other symptoms of the disease are in some way caused by the disease of the supra-renal bodies. They must therefore depend in some way upon an interference with the obscure functions of those organs. It is, however, as yet unexplained why such conditions as injury, hæmorrhage, lardaceous degeneration, cancerous disease, the growth of various kinds of tumours, even tubercular disease in a tolerably advanced stage, do not produce the same results as the chronic diffuse degenerative change we have described; or why these symptoms should not occur when the organs are wasted in an extreme degree, or even, as it would seem, quite absent.*

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* Klebs enumerates 159 well-marked cases (omiting all doubtful or imperfectly observed) and finds that fifty-five were decidedly non-tubercular (taking this word in its widest acceptation), eighty-six were combined with tubercle either in the same organ or elsewhere, while in eighteen the tubercular character was doubtful. Of the non-tubercular cases, eleven only were without pigmentation of the skin, while this was the case with thirty out of the eighty-six tubercular cases, which seems to show that the non-tubercular disease has a more powerful influence in producing the skin affection than has the tubercular. This may be explained by the consideration that possibly the tubercular cases were in an earlier stage of disease.

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* The following references may be made to the chief sources of information on Addison's disease. Addison on "Disease of the Supra-renal Capsules," 1855. Wilks, "Guy's Hospital Reports," ser. III. vol. viii. 1862. Greenhow, "Trans. Path. Soc." vol. xvii. p. 310 (large collection of cases). Virchow, "Die Krankh. Geschw.," vol. ii. p. 688. Klebs, "Path. Anat.," p. 585, Berlin, 1870. Martineau, "De la Maladie d'Addison," Paris, 1864. Averbeck, "Die Addison'sche Krankheit," Erlangen, 1869 (the two latter works we have not had access to). Besides numerous cases in "Trans. Path. Soc.," from vol. viii. onwards, by Baly, Hutchinson, Wilks, Greenhow, Murchison, Bristowe, and many others.

MORBID ANATOMY OF THE URINARY APPARATUS.

CHAPTER XXXVIII.

ABNORMAL CONDITIONS OF THE KIDNEYS.

Congenital Anomalies.—The kidneys are very rarely absent entirely; but it is not uncommon to find one only. In this case Rokitansky makes a distinction between the unsymmetrical and the solitary kidney. The former has its usual position and shape, and is only larger than natural. The solitary kidney is produced by the more or less complete fusion of the two organs together; in its lowest degree it constitutes the horse-shoe kidney, the lower parts of each being connected by a band of renal substance passing across the vertebral column. In the highest degree, there is only a single disc-like kidney, lying in the median line, and situated much lower down, at the promontory, or in the concavity of the sacrum.

When one kidney is absent, it is far more often the left than the right, in the proportion of 7 to 2. It may be absent altogether as well as the ureter and corresponding renal vessels, but sometimes a small mass of fibrous tissue with a renal vein and artery is seen surrounding a comparatively normal pelvis and ureter leading into the bladder.

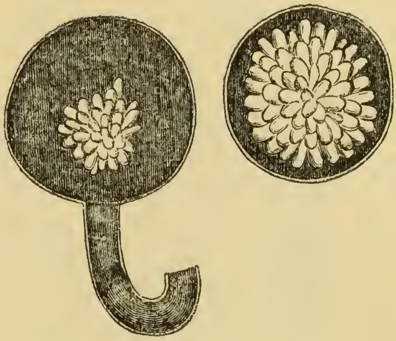
Anomalies of Position.—The kidney is sometimes found much lower than its usual situation, either at the level of the promontory of the sacrum or quite in the pelvis, sometimes in the middle line. In such cases it is usually abnormal in form. This anomaly is doubtless congenital. The moveable kidney lacks its usual attachment to the abdominal wall, or is suspended by a sort of mesentery formed by its peritoneal covering. The vessels and ureter are usually normal. It occurs more frequently in women than in men, and more often on the right side than the left.

Hypertrophy and Atrophy.—Hypertrophy of one kidney is always observed when the other is destroyed or its function interfered with. No simultaneous hypertrophy of both kidneys, without disease, has been observed.

Atrophy is the ultimate consequence of several diseases, which will be spoken of hereafter, but also occurs in the course of senile decay, more constantly than atrophy of almost any other organ except the spleen. This condition appears to be always connected with thickening and obstruction of the arteries. This senile atrophy of the kidney is extremely difficult to distinguish from granular atrophy dependent on disease.

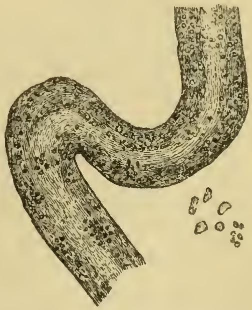
Hyperœmia.—This condition of the kidney is of frequent occurrence, but is almost always the result of some prior general affection, as of obstructive disease of the heart, or the arrest of the cutaneous transpiration. We shall describe an extreme case, to convey an idea of the state of congestion that may often be inferred on good ground to exist, and shall hereafter refer to it as the commencement of other affections. The kidney is enlarged, its weight often doubled; it is of dark red colour, and drips with blood when cut into. The cortical substance, the medullary cones, the mucous lining of the calyces and pelvis, are all much congested. The former is somewhat softened, of a dark-red colour, and presents in many cases small dark-red spots, which are the result of hæmorrhage into and between the tubes. The Malpighian tufts are also distinctly seen as minute, reddish, sub-transparent grains, prominent on the cut surface. In the medullary cones, the congested vessels form long dark-red streaks. A somewhat turbid sanguineous fluid is contained in the injected calyces and pelvis. The capsule, if the hyperæmia has befallen a healthy kidney, can be peeled off readily. Microscopy shows the Malpighian and other capillaries loaded with blood, extravasation sometimes into the capsule of the former, and often into the channel of the tubes. In a typical case no other alteration would be visible, but it is scarcely conceivable that the hyperæmia should proceed to any great degree without exudation of fibrinous fluid taking place, which is then seen having coagulated in the tubes, forming casts of their interior, and

FIG. 151.



Hæmorrhage into Malpig. capsules compressing the tufts.

FIG. 152.

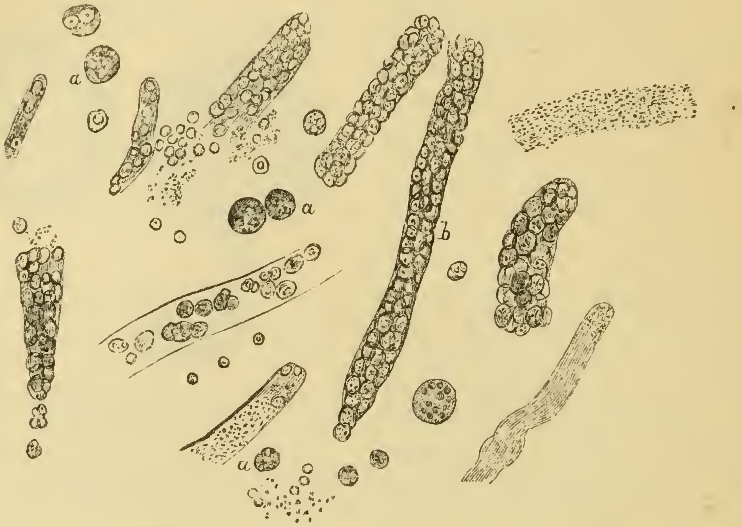


Tube containing some yellow granules, the remains of extravasated blood.

consisting of a granular or homogeneous material entangling blood globules, and often some detached particles of epithelium.

Acute Hyperæmia.—A kidney in the state of hyperæmia, which we have above described, or somewhat approaching to it, with fibrinous exudation in the tubes, is considered by Frerichs as in the first stage of Bright's disease; we greatly doubt if this is generally the case, and cannot look upon hyperæmia as a *necessary* element in the morbid process. Dr. Johnson considers the same condition as the result of acute desquamative nephritis, and believes that complete recovery may not unfrequently take place. In this we quite agree with him, and so, we think, would most observers. He differs, therefore, from Frerichs, in not considering the hyperæmic condition as constituting the first stage of Bright's disease. The urine produced by kidneys in this state is very characteristic; it is of a rather deep, smoky-red colour, with a copious dark-reddish deposit, consisting of blood corpuscles, renal epithelium, and fibrinous casts entangling more or less of the latter. Sometimes corpuscles are present which resemble those of pus, and like them have compound nuclei. The appearance of this deposit will be best understood by reference to the accompanying figure. The hæmatine of the blood globules exudes from them, and gives the peculiar red colour to the urine, which is at the same time scanty and loaded with albumen. For some time after the hæmorrhage has ceased, casts of the tubes, which become, however, more delicate and pale, traces of renal epithelium, and albumen are still discoverable in the urine, until recovery is complete.

Fig. 153.



Drawing of red deposit from urine in intense renal hyperæmia.

We have thus noticed the hyperæmic, or acute inflammatory disease of the kidney, because, though not believing it ourselves to belong to the truly degenerative processes, it produces symptoms in some measure similar, and is regarded by one of the best authorities as always taking the initiative in a more or less marked manner. It may of course give rise to a true degeneration, and apparently pass into it, but there is not more connection, we believe, between the two, than between an attack of bronchitis or pneumonia and succeeding pulmonary phthisis.

A condition of intense hyperæmia is seen in the kidney affection which sometimes accompanies the puerperal state, and appears to cause puerperal convulsions, as is well shown in Plate 6 of Dr. Dickinson's work.* If this pass into chronic disease, which is doubtful, it appears to produce the contracted granular kidney of Bright's disease.

Varieties of Hyperœmia.—This may, of course, be either partial or general, and, as shown before, either active (arterial), or passive (venous). General hyperæmia, proceeding from active congestion, has the typical appearance above described. Partial hyperæmia from the same cause is known by chiefly affecting the surface and the cortical substance of the kidney, which are injected and marked with hæmorrhagic spots; the more noticeable as this part is normally paler than the pyramidal portion. If the kidney be partially injected after death, the injection will be found to fill the cortical portion first, leaving the pyramids uncoloured.

Partial hyperæmia from venous congestion will first affect the pyramids, and these parts will be even more than normally darker than the cortex. Such a condition is seen when the kidneys have suffered venous congestion from some cause which has operated not long before death. The colour is, as usually the case with venous congestion, darker and more purple than that of active or arterial congestion.

Chronic congestion is the result of passive, not active, congestion, and this is most frequently produced by valvular disease of the heart, or obstructions in the pulmonary circulation. In an early stage, when the obstruction has existed not many months, the kidney is found enlarged, the substance hard, dense, and rigid; the colour darkened or purplish, and not due only to fulness of the capillaries but to staining of the tissue, since washing does not remove it. The capsule is easily removed, and the surface smooth. Microscopic examination shows little that is characteristic except general vascular fulness, and apparently some thickening of the walls of the tubes and the fibrous tissue generally, but no interstitial production of new tissue. The epithelium of the tubes may be quite natural. In other cases the chronic congestion is combined with degeneration, and the kidney substance appears mottled or pale when the blood is washed away. If the causes continue to operate the degeneration passes into atrophy, and the whole gland diminishes in bulk; the wasting being most obvious in the cortical portion. In this stage the capsule is thickened or splits into layers on removal; it is unduly adherent in parts, or generally leaves a somewhat rough surface with fine uniform elevations, or what is called minutely granular. The colour of the surface and the substance is a uniform dark-red, the consistency hard and tough. The last stage of chronic congestion from heart disease much resembles cirrhotic or granular disease of the kidney, to be hereafter described, but is distinguished by the characters already given. In the same way, as was shown before, the liver in a state of chronic congestion may approach in some degree to the condition of cirrhosis.

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* On the "Pathology and Treatment of Albuminuria." London, 1868.

Hœmaturia.—We may here notice somewhat further the circumstance of renal hæmorrhage. It may occur as an endemic, and, to judge from the absence of bad effects, scarcely serious phenomenon. Certain individuals are subject to attacks of intermittent hæmaturia. Many of these are probably instances of what is called Hæmatinuria, the urine being coloured by blood, but not containing red corpuscles. Turpentine and cantharides have not unfrequently caused renal hæmorrhage; they act as irritants upon the organ, and produce a state of congestion. Malignant fevers, purpura, and scurvy, are not uncommonly attended with hæmaturia, and there is good reason to believe that in most cases the blood comes from the kidney. In some of these affections, characterized by a fluid condition of the blood, the casts of the tubes would probably be absent, or very imperfectly formed. Hæmorrhagic infarctions or blocks in the kidneys are also attended with hæmorrhage. Blows on the loins are another cause of renal hæmorrhage. A calculus lodged in the calyces or pelvis of the kidney is often the cause of hæmorrhage, which, though taking place from the kidney, and perhaps being very abundant, is distinguishable from the preceding forms, as well shown by Dr. Johnson, by the circumstance that there are no casts of the tubes. These cannot be formed, as the blood not being effused from the renal tissue, but from the mucous membrane, does not traverse their channels. Cancerous disease of the kidney is sometimes attended with bleeding, but this can scarcely be distinguished from that which is the result of the irritation of a calculus. Blood globules, when retained in the tubes, sometimes undergo change into small yellow corpuscles, very much like those which are often found in the spleen. It is necessary to be aware of this circumstance, as otherwise the observer might suppose that biliary matter was present.

Anœmia.—General anæmia does not seem to affect particularly the condition of the kidney, at least we have notes of one case of extreme anæmia the result of menorrhagia, and which at last terminated fatally, in which, although the kidneys were small, the epithelium of the tubes was very perfectly formed. Mr. Simon notices, however, a condition of atrophy of the epithelium, resulting from the obstruction of an arterial branch by atheromatous and fibrinous matter, which would of course produce a local anæmia. In that which is regarded by several as the second stage of renal degeneration, the kidney, though much enlarged, is often remarkably pale. This anæmia, however, is not attended with atrophy, and is itself, probably, in part the result of pressure exercised on the intertubular plexus. Anæmia is the most constant result of obstruction of the small vessels by waxy degeneration.

SUPPURATION IN THE KIDNEY.

Pus may be formed in the kidney under two conditions, (1) from suppurative nephritis, (2) as pyæmic abscess.

(1) *Suppurative nephritis*, analogous to common inflammation of other parts, and like it often passing into suppuration, is not a very common disease; it constitutes what is generally known as renal abscess. It is most frequently seen as the result of the irritation of calculi in the kidney, or of inflammation of the bladder, which has either spread up along the ureters, or directly attacked the kidney. Hence this form of disease has been called Surgical Kidney. Blows on the loins are also mentioned by Drs. Prout and Johnson as causes of nephritis. Whatever be the cause, the general characters of the inflammatory action will not differ materially, provided it is true nephritis that we are dealing with, and that it passes on to the stage of suppuration. Nephritis, unattended with the formation of pus, would probably be undistinguishable from the condition of hyperæmia above described.

The kidneys are usually enlarged, and in some parts very much injected; in others, the red surface has numerous whitish spots, or patches, appearing through it; these are seen in sections to be the ends of long striæ, which commence at the base of the medullary cones, and extend to the surface of the organ. Between them are interposed streaks of congestion. There is often nothing remarkable in the medullary cones. The minute changes affect both the tubes and the interstitial stroma. In a case which we examined, the cortical tubes were quite infarcted with their epithelium; in the whitish portions this was especially accumulated, and altered so that it resembled a mass of nuclear particles; the tubes also were not clearly seen; they were doubtless so distended and crowded together that their outlines were lost. In some parts the basement membrane of the tubes was gone, and the contents appeared as a naked strand of nuclei and granular matter, part of which became dispersed between the tubes, and made the mass quite uniform. This mass of altered renal structure was evidently on the point of fusing down into fluid pus. The Malpighian tufts appeared healthy. The medullary tubes were also infarcted, some of them very much, and were opaque, as if containing finely-divided oily matter. The urine in these cases is found to contain cylindrical masses of pus cells, which have evidently been formed in the renal tubes; this sign, while it lasts, serves to distinguish suppuration in the kidney from that taking place in the bladder, as in the latter case the pus corpuscles form only shapeless masses.

The changes in the interstitial substance are not less marked in many cases, and more specially characteristic of this disease. The tubes are seen widely separated, and the intertubular stroma infiltrated by collections of leucocytes or pus corpuscles, such as are seen in all inflammations, and the accumulation of which constitutes pus.* If the walls of the tubes and fibrous structures are softened and broken down, an abscess results, but this is not always the case; as the inflammatory process, when it starts from affection of the bladder or urethra, seems to become chronic, or to occur in repeated attacks. The evidence of this after death is the irregular wasting and puckering of the surface, the adhesion of the capsule, and beyond the capsule of the fatty and connective tissue round the gland; which can sometimes hardly be removed (Perinephritis). This special participation of the external envelopes is explained by a peculiar fact in the distribution of the morbid process, that even when it starts from the pelvis, the next most prominent seat of the disease is the surface; the intervening part being marked merely by the whitish striation mentioned above. On the surface are seen yellow elevations composed of foci of incipient suppuration, which have a singular resemblance to pustules of the skin. With these is active inflammation (though not suppuration) of the capsule and surrounding tissue. The origin of the pus cells in nephritis may of course be explained on any of the three hypotheses before mentioned (p. 87), but the probability seems great that here, at least, they are emigrated leucocytes.

The mucous membrane of the calyces and pelvis, especially when a calculus is lodged in these cavities, is softened and inflamed, and secretes a purulent fluid. The ureter is generally comparatively unaffected, but the bladder or urethra, or both, are inflamed in most of these cases; and the urine is usually in a state of alkaline fermentation.

This fact, combined with certain appearances in the suppurative kidney, has led Klebs to suppose that the secondary inflammation of the kidney depends on the ascent of bacteria from the pelvis into the uriniferous tubes and kidney substance; whence he calls the disease Pyelonephritis, or Parasitic Nephritis. In sections of a kidney in this disease, tubes and stroma, he says, often contain collections of brilliant granules, distinguished from fat by their insolubility in acids, alkalies, or ether. These are the bacteria, and the same are contained in alkaline putrefying urine. We believe that such granules occur, and that they are really bacteria; but how far they are to be regarded as the *cause* of disease is yet uncertain. Dr. Dickinson also attributes the inflammation to the absorption of septic matter from the urine ("Med.-Chir. Trans.," vol. lvi. p. 223).

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* See a very good figure in "Trans. Path. Soc.," vol. xxiv. plate 4, by Dr. Goodhart.

The extension of renal abscesses may go on until the whole organ is converted into a mere pouch of pus; in this case, or even before the organ is quite destroyed, the abscess may make its way by the usual process of absorption, penetrating through surrounding indurated tissue, and evacuate its contents in either of the following situations:—(1) Externally into the lumbar region; (2) into the cavity of the peritoneum; (3) into the ascending or descending colon, or into the duodenum; (4) into the bronchi after perforation of the diaphragm. Acute inflammation of the kidney may become chronic, or the inflammation may have a chronic character from the outset; its results may be suppuration or induration, and consecutive atrophy of the organ.

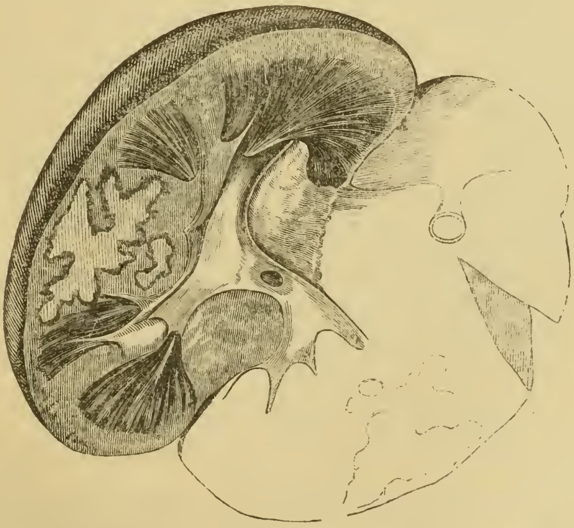
Suppuration in the tissue surrounding the kidney, or *perinephritis*, is generally the consequence of wounds or injuries, or of the extension of inflammation from neighbouring parts.

(2) *Pyæmic abscesses*, if they occur in the kidney, are similar to those in other organs, and do not call for any special remark.

Two conditions which may be confounded with them must, however, be mentioned:—Infarctions, or fibrinous blocks, and capillary embolisms.

Infarctions or Blocks.—These lesions correspond generally to those of the spleen, and, like them, depend upon the arrest of circulation in a portion of the gland; the cause being obstruction

FIG. 154.



Blocks in a granular kidney. The situation of the patch is marked by the irregular outline, which was a deep red.

of the arterial branch. This obstruction is generally occasioned by embolism, a fragment of vegetation from a cardiac valve, or some coagulum or atheromatous matter from the heart or aorta. Hence they are especially common in aortic disease accompanied by vegetations on the valves, and in acute endocarditis. Sometimes no embolus can be traced, and there appears to have been spontaneous thrombosis in the artery. This is especially likely to occur in fevers, as in typhoid. The blocks in the kidney have a less distinctly conical shape than those in the spleen, the vascular distribution being different, but still this shape is seen in smaller blocks. Recent infarctions are sometimes dark red, hæmorrhagic in appearance, dense, and, if near the surface, somewhat elevated above it, but we do not very often see them in this stage, and possibly some are anæmic from the first. Generally some degenerative change has set in; then we find a tough yellow mass of "fibrinous" appearance, bordered by a very bright belt of scarlet injection (Fig. 154), caused by collateral hyperæmia. Later on, the necrosis of the central portions continues, and the surrounding inflammation results in the production of fibrous tissue. Ultimately absorption of the central portion takes place, and nothing but a mass of newly-formed fibrous tissue remains, which, as is usually the case with such tissue, suffers contraction till a puckered cicatrix only is left.[*] This change explains the irregular form of many kidneys in cases of heart disease. The microscopical examination of blocks in an early stage shows the natural structure of the kidney much engorged, and with hæmorrhagic and inflammatory products in the tubules, hence this is one cause of renal hæmorrhage. At a later period little structure can be made out in the central portions which chiefly consist of granular amorphous material like degenerated fibrin. In the peripheral portions new growth of nucleated connective tissue is observed. Pyæmic blocks may sometimes be mistaken for the infarctions just described, but in them, though there may be vascular obstruction, the inflammatory changes ending in suppuration greatly preponderate.

Capillary embolisms are often regarded as entirely belonging to pyæmia, but do not appear necessarily to do so. They occur generally, if not always, in acute endocarditis. We there often find the surface of the kidney covered with ecchymotic spots and with small blocks going a little way into the substance. Similar blocks may be seen in the interior of the kidney. Both parts show a vivid injection round the blocks which are yellowish in the centre. These appearances depend upon obstruction of capillaries or minute arteries; and in what way the obstruction produces hæmorrhage is shown on page 116. Cohnheim objects to the term capillary embolism, contending that small arteries are always obstructed. This is doubtless, in strictness of speech, true; but the phenomena are certainly different from those of obstruction of larger arterial branches. This process, though not necessarily pyæmia, may be a part of one form of that disease. (See page 349.)

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[*] The actual time occupied in these changes is uncertain, but the following observation made by Dr. Herman Weber of a case where embolic blocks existed in both kidneys, gives an approximate date. That in the right, which was diagnosed as having occurred thirty-eight days before death, was already shrunk below the level of the surrounding tissue, while those in the left kidney, having occurred between a week and a fortnight later, were still slightly prominent above the level of the surrounding tissue, but without the elevated red margin seen in still more recent cases. ("Trans. Path. Soc.," vol. xvi. p. 166.)

Simple Degenerations of the Kidney.—Three well-marked conditions may be called degeneration:—(1) Granular degeneration of the epithelium, seen in acute febrile diseases, concurrently with a similar affection of the liver; (2) Fatty degeneration of the epithelium; (3) Lardaceous degeneration, affecting chiefly the blood-vessels.

Fibroid degeneration or cirrhosis will be described under a distinct head.

Granular or Parenchymatous Degeneration.—The kidney is enlarged, especially the cortical portion, smooth, and generally pale, though perhaps with some spots of injection or ecchymosis. The cortical portion has a peculiar opacity and whitish clayey colour (but is less opaque than in some cases of Bright's disease); it is soft, and its appearance has been described as "coarse-grained." The pyramids appear over-vascular but otherwise natural, so that there is a striking contrast between the two parts. The enlargement of the cortical portion depends upon the condition known as "cloudy swelling" of the epithelial elements, characterized by positive increase in size, and by the great abundance of dark granules in their substance. The convoluted tubes will hence appear filled up with opaque masses, but do not necessarily contain any abnormal elements or substance. As the epithelium of the cortical tubes is normally somewhat granular, and often slightly fatty, some experience is needed to recognize this condition, but in general the dark, opaque appearance of the tubes is sufficiently distinctive (see Fig. 156). The epithelium of the straight tubes in the pyramids (obtained by squeezing till a milky drop appears on the papilla) is usually quite translucent and clear. No other changes are constantly present.

This form of disease is met with in the same class of diseases as produce the similar condition of the liver (p. 628), and probably occasions (though how we do not know) the albuminuria so often present in these diseases. It is thought by some pathologists to be the first stage of one of the varieties of Bright's disease, or even to be in fact a mild or acute form of that disease. The condition must, however, in most cases be transitory; and if chronic disease be established, other features will be added, which alter the pathological type. Phosphorus poisoning has been found to produce a similar degeneration of the renal epithelium. Scarlatina is also commonly regarded as producing a typical form of this degeneration; but the type of kidney disease thus produced is not always the same.

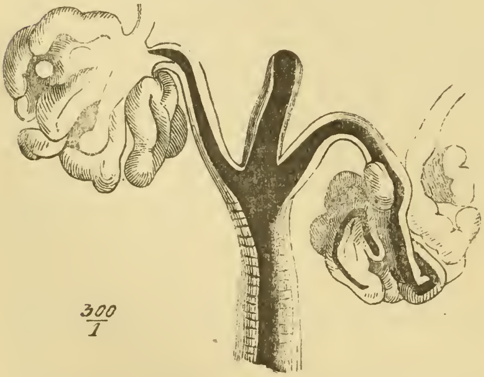
Fatty Degeneration.—This condition is very common, so much so that it can be hardly called a disease. Dr. Dickinson found out of sixty-eight healthy kidneys twenty-five in which the epithelium contained distinct fat globules, while in a still larger number the epithelium was granular. The renal epithelium of dogs and cats is also commonly fatty, as was shown by Mr. Simon. When fatty degeneration occurs in a healthy kidney, or one not seriously diseased, it gives a yellow colour to the cortex which is chiefly seen in a belt just outside the bases of the pyramids, where they form a transition to cortical substance. If more advanced, the change may extend over the whole of the cortical portion, but the epithelium of the straight tubes is seldom or never fatty. It is doubtful how far fatty degeneration, taken alone, can be said to constitute a disease of the kidneys.

In extreme cases the fatty change is found to involve not only the epithelium, but the stroma, the capillary blood-vessels, and the Malpighian tufts.

The extreme cases of uncomplicated fatty kidney are met with chiefly accompanying pulmonary phthisis, but are not to be confounded with those in which there is lardaceous disease also. Fatty degeneration is, however, an accompaniment of several important morbid processes, and is itself the outcome of the granular degeneration just described.

Lardaceous Degeneration.—This disease as it affects the kidney presents certain peculiarities important to notice. It occurs in two tolerably distinct forms. In the first, which is by far the commoner, the kidney is much enlarged, smooth, pale, anæmic, the surface and cortical portion mottled with translucent and opaque yellowish patches, the substance firm and elastic. The addition of iodine produces both a general yellow colour and an orange or brown tint in small specks scattered through the cortex, which are in fact enlarged and degenerated Malpighian glomeruli, while the smaller arteries are sometimes stained in the same way. In the second form of the disease the kidney is smaller than the normal, its substance is

Fig. 155.



Malpighian tufts, and a small artery of the kidney affected with lardaceous degeneration. (After Rindfleisch.)

very generally translucent, as if soaked in wax, and of a yellowish colour, while the cortex is diminished. Iodine produces the most marked change in the pyramids, which, especially the papillæ, are stained of a dark brown. The same colour is seen on the surface of the pelvis, and sometimes even in the ureters and the mucous membrane of the bladder. The first form shows the manner in which the disease first attacks the kidney, through the small arteries and glomeruli; while it is only in cases of long standing that we find the pyramids so much affected as in the second form. The peculiar distribution of the infiltration in the latter suggests a transference of material from the cortex to the papillæ. It is only seen in cases of very advanced disease, and the ordinary appearance of the disease is that first described.

Microscopical examination shows in the glomeruli and small arteries the changes represented in Fig. 155, where it will be noticed that the injection has filled a part only of the glomerulus, an evidence of the most important fact in this morbid condition that it limits the supply of blood to the organ. This sufficiently explains the anæmic condition of a waxy kidney. No similar degeneration is usually seen in the renal epithelium, except in very advanced cases, when the epithelium of the straight tubes and the contents of the tubes—*i.e.*, hyaline matter and altered epithelium, sometimes undergo such a change as to be stained by iodine. But there are nearly always other changes associated with the lardaceous degeneration of the vascular parts—viz., changes in the tubes identical with those in one form of Bright's disease, tubular nephritis, and changes in the interstitial stroma, which are characteristic of those seen in the other or cirrhotic form of that disease. The discussion of these mixed types of disease must be deferred.

The causes of waxy degeneration have already been discussed; there is generally nothing to explain why it should specially affect the kidney.

MORBUS BRIGHTII.

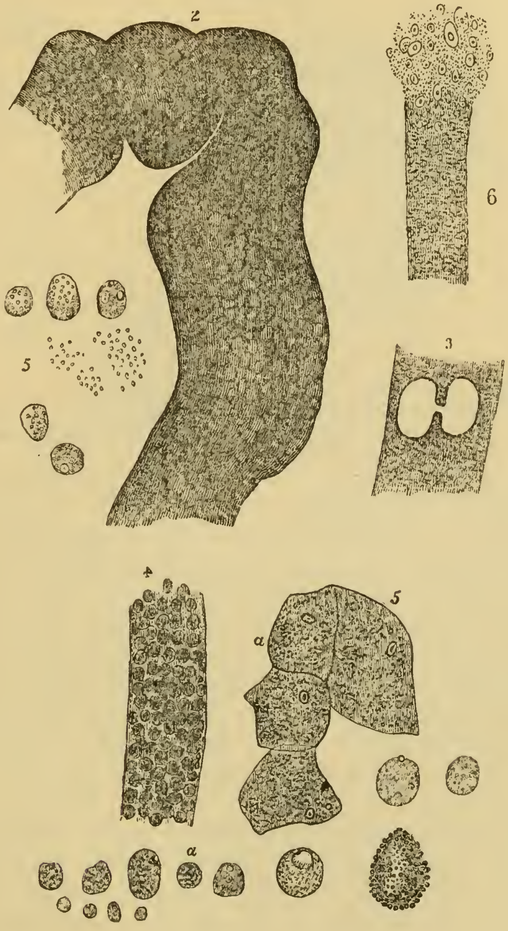
Degenerative Disease of the Kidney; Desquamative and Non-desquamative Nephritis; Subacute Inflammation of the Kidney.—These names refer to an extremely common and important disease or diseases of the kidney, whose main features were discovered by Dr. Bright, but whose real nature is still a matter of doubt. In calling it a degenerative disease, we have expressed our own opinion respecting it, which coincides very closely with that held by the late Dr. Prout. Dr. Bright (according to Dr. Wilks) distinguished two forms of disease with two corresponding conditions of the kidney, and these in their broad features represent the two types of disease which we shall describe. The first is a comparatively acute disease, with a marked beginning, and definite symptoms of which dropsy is always one; the other, a chronic but exceedingly insidious disease, often not discovered till shortly before and sometimes not till after death; generally, therefore, without having given rise to the symptoms of the more acute disease, and sometimes quite unaccompanied by dropsy. The kidneys, in cases of the former disease, are usually found enlarged, especially the cortex, pale, smooth, without any abnormal adhesion of the capsule; it has hence been called the large, smooth kidney. In the second form of disease, the kidney is usually found small, wasted, especially in its cortical portion, the surface rough, granular, the capsule adherent; this has hence been called the contracting granular kidney.

Passing from the naked-eye appearance to the minute anatomy, we find that in the majority of cases the large smooth kidney shows changes in the tubes and their epithelium, partly hyperplastic and partly degenerative (*i.e.*, fatty), without any alteration of the interstitial stroma, and has hence been called tubular nephritis or fatty degeneration. In most cases, also, the small granular kidney presents, with a variable amount of morbid change in the tubes (sometimes hardly any that is well marked), alterations in the interstitial stroma and in the arteries of an hypertrophic kind. It has hence been called interstitial nephritis or cirrhosis of the kidney.

In this summary we leave out of account two forms of kidney disease, which have been separately treated, but which were probably included by Bright under the above two heads, viz., the lardaceous degeneration, formerly unavoidably confounded with the other large, pale, smooth kidneys, and the form of kidney disease dependent upon cardiac obstruction, which constitutes one form of contracted granular kidney. Besides these, there are, however, certain important exceptions, which must be mentioned. In the first place, some examples of "large smooth kidney" show, on microscopical examination, not so much changes in the tubes as pronounced interstitial growth, or the alteration regarded as characteristic of the second form. These cases may, perhaps, be compared to those of large, smooth cirrhosis of the liver formerly described. In the second place, some small kidneys with granular surface show simple wasting, without interstitial growth, and may be regarded perhaps as examples of the same form of disease as usually gives rise to the large, smooth kidney, but in an advanced stage of degeneration. These two forms are, however, comparatively rare; there are also mixed or intermediate forms.

Parenchymatous or Tubular Nephritis; sometimes called Fatty Degeneration.—In this form of renal disease, the kidney is considerably enlarged, even more, sometimes, than in acute hyperæmia; both kidneys have been found weighing twenty-three or even thirty ounces; it is generally pale, sometimes of an opaque greyish-white, with mottling spots or streaks of red; its capsule peels off readily; the surface is smooth, and tolerably uniform, or presents opaque spots, while, rarely, granulations may be just beginning to become prominent. The surface of a section shows the thickness of the cortical substance much increased, and the medullary cones also somewhat enlarged, and markedly striated, so as to resemble a plume of feathers; they are usually more congested than the cortex. The renal tissue in the latter part appears

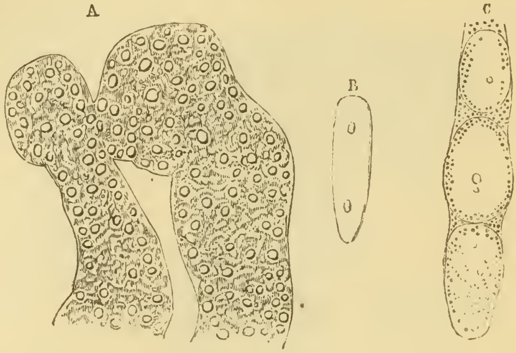
Fig. 156.



2, Cortical tube, infarcted with epithelium, and bulged in a good part of its extent.
3, Cortical tube, containing a dumb-bell crystal of large size.
4, Cortical tube, infarcted by epithelium, at (*a*) below it; some of the separate particles are shown more highly magnified.
5, Bulky epithelium from cortical tube, the group at (*a*) are remarkably enlarged, those below them are more or less fatty.
6, Medullary tube much infarcted, the contents are seen escaping from the upper end.

to be obscured or confused, as if some coagulating fluid had been effused throughout it; it is commonly marked also by indications of commencing granulations similar to those which are seen on the capsular surface. The mucous membrane of the calyces and pelvis is somewhat swollen and reddened. The consistence of the kidney is rather diminished, it is rather flabby and soft. The above description applies to many instances of an early degenerative condition, but there are many others also, in an early stage, which differ in several respects. The size of the organ may not be materially increased, though its structure is evidently altered; its consistence may be dense, firm, and brittle, instead of soft and flabby; its capsule may be firmly adherent, and its colour more approaching the natural. Cysts are sometimes observable on the surface of the organ, but this is no necessary part of the morbid change.

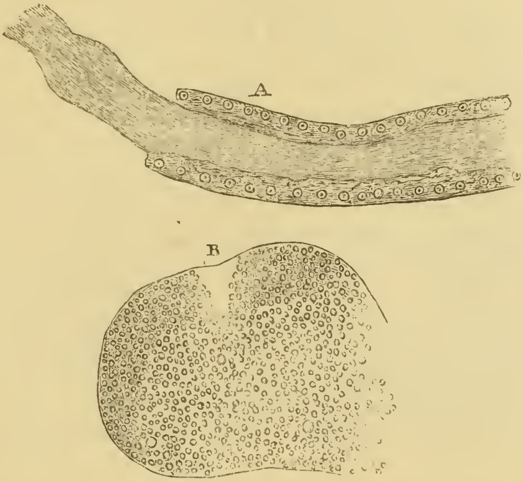
FIG. 157.



A, Cortical tubes, containing a very fatty epithelium.
B, A short homogeneous cast, containing two corpuscles.
C, Portion of a medullary tube, containing three casts, looking much like cysts and oily matter.
The three figures (156 to 158) are intended to illustrate the changes observed in the enlarged form of degenerated kidney.

Histology of Tubular Nephritis.—The epithelium lining the cortical tubuli is increased in bulk; sometimes its particles are more completely formed than usual, and appear more distinct and separate; sometimes their size is greatly exaggerated, so that a few particles cohering together form a large bulky mass; sometimes the tube is filled with a *mélange* of stunted or withered-looking nuclei, granular matter, and ill-formed celloid particles. The central channel of the tube, which should, normally, be about one-third or one-fourth the whole diameter, is much encroached on and even obstructed by this accumulation of epithelium; from the same cause the tube becomes dilated, and hence the bulk of the organ is increased. Cylinders of fibrine, or hyaline matter, also are present in some of the tubes, but not in the great majority, and doubtless aid in increasing the obstruction. The basement membrane of the tube, in some cases, is natural, in some, is decidedly atrophied, and scarcely can be detected. A condition of the epithelium is sometimes observed which is extremely significant, we think, of the nature of the morbid process; it is noticed by Dr. Johnson as peculiar to that form of disease which he denominates non-desquamative nephritis. The epithelium in this does not accumulate and block up the tubes, but appears as a coarsely granular opaque stratum, of the natural width, resting on the basement membrane.

FIG. 158.



A, Tube containing an homogeneous cast, which projects from its broken end. B, Malpighian body, the capsule is filled with oily matter.

Besides the swollen and opaque epithelium, cells are sometimes seen which are regarded as indicating proliferation of this structure and have led to this process being described as a "renal catarrh:" that is to say, detached, spheroidal masses like epithelium, lying in the middle of the tubes. Where there is no epithelium on the wall of the tube they appear to have been formed out of it, though not of necessity indicating actual cell-proliferation. But more generally the adventitious cells are simple exudation- or pus-corpuscles.

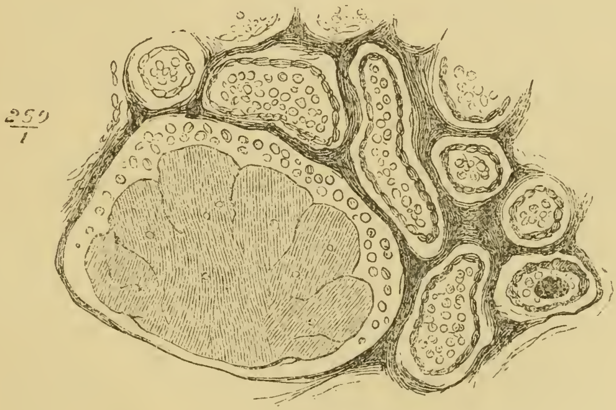
The Malpighian tufts are more opaque than natural, the capillaries being obscured by some material contained in the capsule which is probably exudative, but which some pathologists derive from the very delicate epithelium lining the capsule. In some cases this is so abundant, and so mixed with oily matter, that the capsule becomes notably dilated; in others, the pressure of the refluent fluid from the obstructed tube compresses the tuft into a small space at the bottom of the capsule. Oily matter, in the form of minute dark molecules, and various sized drops, is often present in the epithelium in small, sometimes in considerable quantity; when very abundant, it imparts to the kidney a dead milky or yellowish-white aspect. In such cases the epithelial particles often become so filled with oily molecules as to resemble very closely granule cells. In many cases, however, there is no trace of it, and it is certain that it is not of the essence of the disease. In the more advanced instances of degeneration, before, however, atrophy of the kidney has become decidedly apparent, the cortical tubuli exhibit unequivocal traces of breaking up. Their basement membrane is lost; their epithelium, though still preserving the tubular form, is tending to become a mere detritus, and the commencing appearance of granulations shows that atrophy is taking place in some parts, while others remain prominent and distended by their included cell growth. Microscopic cysts are sometimes imbedded in the cortical tissue, and may tend, in some measure, to increase the size of the kidney, but they are not so numerous as in the more atrophied condition. The medullary tubuli are less affected than the cortical; those near the base of the cones, especially, often are filled by accumulated epithelium, while those nearer the mammillæ are more free, and contain, either some oily matter, or fibrinous casts, or yellow corpuscles, the result of hæmorrhagic effusion. The matrix tissue is little altered in itself, but is in some cases infiltrated here and there with a granulous exudation matter containing a few nuclear corpuscles; these are sometimes elongated and developing fibres. These are the exceptional cases, to which we have already referred.

Morbid Contents of the Tubes.—The uriniferous tubes in this, as in some other morbid conditions of the kidney, contain, beside the altered epithelium, two kinds of products, which deserve special attention, viz., the hyaline or so-called fibrinous cylinders or casts and newly-formed cells or corpuscles. The composition of the former is not positively known; no fibrillation is usually perceptible in them, though it is said that in rare cases some approach to it has been observed, and they seem to have been called fibrinous chiefly because they were regarded as the result of exudation. This view has given rise to the name croupous nephritis, applied to one form of kidney disease by some German pathologists, from its supposed analogy with croup of mucous surfaces and "croupous pneumonia." Rokitansky, on the other hand, regards all hyaline cysts, as formed by the metamorphosis of the renal epithelium by a sort of colloid degeneration; and when the cast and epithelium are found in the same tube (as in Fig. 158), supposes the cast to be the result of metamorphosis of epithelium higher up the tube. This appearance is, however, too general to be entirely explained in this way, and it must be taken as proved, that the hyaline material is in most cases something beside epithelium. Rindfleisch describes this substance in question as a product of the epithelium, especially of the straight tubes. The epithelial cells, he supposes, produce colloid masses in their substance; and these at length become fused into a cylindrical mass, filling up the tube. It is not easy to trace this process; but it must be admitted that occasionally a substance not unlike in appearance the material of the hyaline casts is seen in drops or small masses within the tubes. This may sometimes be seen in fine sections, or in the drop of liquid which is obtained from the straight tubes, by squeezing the papilla. The drops are known from oil drops by the want of refractive power, and hence by not appearing dark or light as the focus is altered; and from any modification of cell forms by their perfect uniformity and fineness of outline. They do not appear to pass into the urine, and are perhaps partly a post-mortem production.

Other authors, as Klebs, believe in two methods of formation. Some casts they believe to be formed by a process resembling secretion from the epithelium; others, as Rokitansky supposes, by metamorphosis of the epithelium. The latter, according to Klebs, are distinguished by their larger size (as they occupy the whole of a tube), and irregular or varicose outline. They do not, he thinks, usually pass into the urine. It must be admitted that the evidence for any one of these modes of origin of the casts, as for their fibrinous nature, falls short of actual demonstration. Undoubted fibrinous solidifying exudation is sometimes seen within the capsule of the Malpighian tuft.

Origin of New Cells.—Very divergent views have been held with respect to the origin of the cells seen within uriniferous tubes. Those which occur in most conditions of inflammation or degeneration, especially in chronic tubular nephritis, are indifferent corpuscles or leucocytes, like those of ordinary inflammations, and taken singly, are indistinguishable from pus cells. They have been regarded as derived from the renal epithelium by proliferation, and hence called catarrhal cells; or, as formed in a similar manner from connective tissue corpuscles; or finally, as white corpuscles emigrated from the blood-vessels. In favour of the former hypo-

FIG. 159.



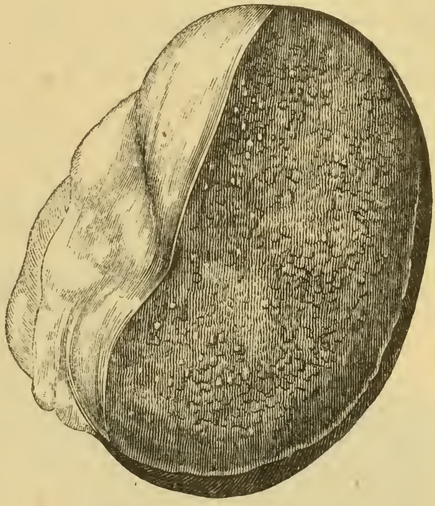
Section of a kidney, showing a Malpighian tuft surrounded by lymphoid corpuscles which have probably found their way through the capillary wall. Similar corpuscles are seen in the surrounding tubes with unaltered epithelium in its natural position. To the right of the figure is one tube with a homogeneous hyaline cylinder cut across.

thesis may be urged the fact that tubes are sometimes found filled with corpuscles, and without any epithelium, as if the latter had become changed into the former; the existence of what are regarded as transitional forms, and finally the alleged analogy of catarrh on mucous surfaces. But since Cohnheim's observations on the emigration of blood corpuscles, the latter origin has become much the most probable, and is particularly supported by such appearances as those in Fig. 159, where we see exudation corpuscles surrounding the Malpighian glomerulus, and further, filling all the surrounding tubes, the epithelium of which is intact. In this case it is clear that the corpuscles lie precisely in the track of matters exuded from the vessels. It is possible that here, as in other cases of inflammation, new cells may be produced from more than one source.

Urine of Parenchymatous or Tubular Nephritis.—The urine produced by kidneys in the foregoing condition is almost invariably diminished in quantity, generally paler than natural, of somewhat altered smell, and deposits a whitish sediment, consisting of fibrinous casts, renal epithelium, and vesical scaly particles. Its specific gravity, which in the earlier stages may be above, tends to fall more and more below, the standard of health. When oil drops appear in the epithelium or in the casts, it is, of course, a sign that oil is deposited in the kidney, but it is by no means certain that this particular change is of any moment. Albumen is constantly present, except in a few rare cases, where it disappears for a short time; its quantity varies considerably, being, apparently, much more influenced by other circumstances than by the period of the disease, but always more than in the other form of the disease.

Granular Atrophy of the Kidney; Cirrhosis of the Kidney; Chronic Interstitial Nephritis.—The next condition of the kidney which we shall describe is often regarded as a further stage of the preceding; this is, we believe, sometimes the case, but not by any means necessarily or universally. The organ is greatly atrophied, and evidently contracted; its surface is covered with irregular prominences, the so-called granulations. Its consistence is considerably increased, so that the structure has, as Frerichs says, a kind of leathery toughness; this is more marked in proportion to the atrophy. The colour is decidedly less pale than in the former variety, but has generally the following peculiarities. Pale yellowish granulations covering the surface are separated by tissue which is somewhat more vascular and less yellow. The granulations correspond to the natural divisions of groups of tubules which may be traced dimly in the healthy organism. Sometimes the colour is much more uniformly red, but this colour depends rather upon passive hyperæmia than on the actual colour of the tissue, and is more often seen in cases of wasting dependent upon venous congestion than in the true "granular kidney." On section the colour is usually pale yellowish and mottled, but the pyramids somewhat more vascular. This condition presents, especially externally, an unmistakable analogy with cirrhosis of the liver; the granulations corresponding to the yellow projecting masses of degenerated tissue. At the same time the intervening tissue in the kidney is less vascular; as might be expected, considering the double system of afferent vessels in the liver. Hence this has been called the cirrhotic form of kidney disease. The capsule is always very adherent, but it is worth noticing that, sometimes, when it is thickened, a layer of it may be peeled off, leaving behind an apparently smooth surface. The cortical part is most affected by the atrophy, being reduced, in extreme cases, to a layer two or three lines in thickness; the medullary cones suffer in a less degree. The external form is often remarkably lobulated, reminding one of that of the fœtal kidney.

Fig. 16[illegible].

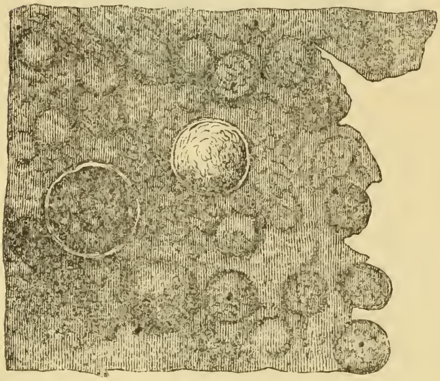


Drawing of atrophied kidney.

Microscopic Appearances.—The renal tissue is found in this form of kidney to have perished extensively, and this generally in proportion to the shrinking of their size. In extreme cases, one may scarce find in a section anything of the secreting structure except mere granular *débris*, some of these, perhaps, still preserving the contour of the original tube, but the greater part constituting an indefinite shapeless mass. In other, less advanced, cases, and in some parts, indeed, of all, the tubes are still discernible: they are irregularly distended, and opaque with granular contents, which have well-nigh, or perhaps completely, blocked up their canals. Oily molecules, sometimes accumulated in considerable quantity, lie here and there amid the granular matter, and increase the opacity greatly. The granulations are made up of the infarcted convolutions of tubes, and are the parts in which most traces of the natural structure still persist; they remain prominent, because the intervening parts have perished and shrunk in. The atrophied tubes and Malpighian bodies will then be found separated by what is the most important structure in the cirrhotic kidney, a nucleated or small-celled tissue, which in some parts shows definite fibrous organization. This is evidently a species of newly-formed connective tissue, and, as such, possesses the power of contraction usually belonging to such tissue. It is to its shrinking that the irregularities of the outline in the granular kidney are due. The changes are, in general, similar to those seen in other organs, as a consequence of "fibroid degeneration" or fibroid substitution; and it is probable that the small-celled infiltration and atrophy go hand in hand, rather than that there is first a stage of cellular infiltration followed by a stage of atrophy, as the more recent German writers describe. This nuclear tissue is found very unevenly distributed through the kidney; chiefly in the cortex, and often passing in from the periphery, as Dr. Dickinson has especially shown. We may often, also, trace a close connection with the outer coat of the arteries (Fig. 162), a point to which Sir W. Gull and Dr. Sutton attach much importance. It is also especially abundant round the capsules of the Malpighian tufts, and this more particularly in what appear to be early stages of the disease.*

We have observed sometimes a single tube with its homogeneous membrane denuded by separation of the epithelium, as noticed by Dr. Johnson, but in sections the appearance of an empty tube is liable to some fallacy, since the epithelial contents may have fallen out in the process of preparation. We have also seen what we think has not yet been described, a kind of thickening, or hypertrophy of this membrane, which has seemed to us to result from the atrophy of the epithelium, and its fusion into an homogeneous layer on the inner surface of the tube. The walls of the tubes and the stroma generally are often uniformly thickened. The Malpighian tufts, in consequence of the general collapse, appear closer together; a few of them remain tolerably healthy, others are compressed and shrunken; often the capsule is filled, to a greater or less extent, with an oily-looking matter, or the capillaries are obscured by fibrinous exudation. A very important and significant alteration has been observed in the condition of the small arteries by Dr. Johnson; he finds their coats considerably thickened (Fig. 162), both the inner of longitudinal and the outer of circular fibres, and he regards this thickening as an instance of true hypertrophy induced by the increased pressure exerted upon their parietes by the retardation of the circulation through the inter-

Fig. 161.



Cortical part of a very granular kidney, containing very numerous microscopic cysts. The tubes are very much degenerated and broken up. Two Malp. bodies are shown.

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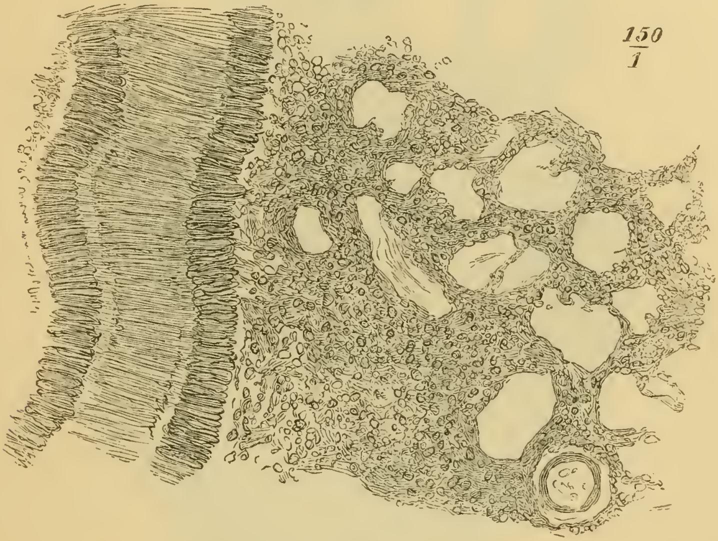
* Traube distinguishes two forms of granular kidney; that in which the small-celled infiltration is especially abundant round the Malpighian capsules, and that in which it occurs round the intertubular capillaries.

tubular venous plexus. There is also often general thickening of all the coats, rigidity, atheroma, and calcareous degeneration of the arterial wall, with diminution of its calibre, as has been well figured by Dr. Lionel Beale. No particular change is observable in the before-mentioned capillaries, or in the veins, except that the latter often contain firm coagula of blood, which are more or less closely adherent to their walls, so as sometimes to constitute actual thrombosis.

Fig. 162.



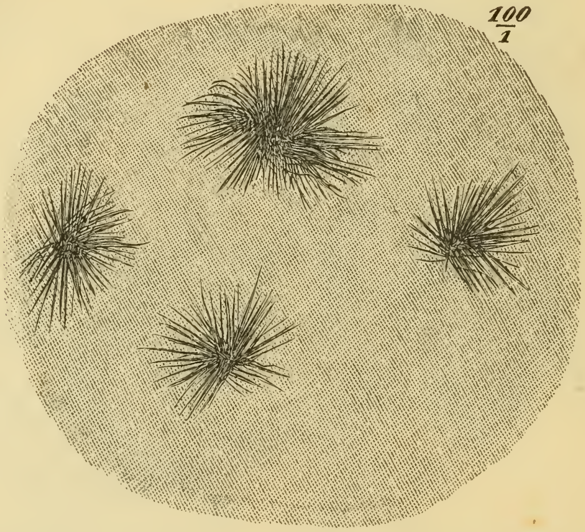
Section of kidney in advanced granular degeneration; showing nucleated fibrous tissue diffused between the tubes, the contents of which have been removed by washing, except in one instance when the epithelium is seen unchanged, but the cavity contains some corpuscles. A small arterial branch with hypertrophied muscular coat is also shown.

(From an original preparation.)

Large Form of Interstitial Nephritis.—In some cases the anatomical changes just described are met with in kidneys which are large, smooth, and pale, in which the cortex is enlarged, and the capsule easily removed, and which are, in short, not to be distinguished by the naked eye from kidneys affected with parenchymatous nephritis. These constitute, accordingly, a certain proportion of the "large, smooth kidneys" of Bright. The secreting structure is in such cases often little altered; a fact which has led some writers to regard this as the necessary first stage of the contracted granular kidney. There is, however, no proof that all contracted kidneys have passed through this stage, and we may again refer to the parallel of liver diseases; comparing this form of kidney to the large, smooth form of cirrhosis (*smooth interstitial Hepatitis*, p. 623). In persons who die of kidney disease this form is certainly rare.

Gouty Deposits in the Kidney.—In contracted granular kidneys it is not uncommon to find, chiefly in the pyramids, white lines and specks of mineral matter which, on microscopical examination, are found to consist of masses of urate of soda, like those of ordinary gouty deposits. These masses are imbedded in the thickened fibroid matrix, not contained in the tubes; though under certain circumstances masses of urates may be found in the latter situation. The deposit is sometimes granular, but more often in the form of stellate bunches of acicular crystals, as shown in Fig. 163. This condition often accompanies general gout; sometimes when no symptoms of gout have occurred, similar deposits will be found in the articular cartilages of the great toe. Sometimes it is alleged to be the solitary gouty deposit in the body. To make a special form of kidney disease—arthritic nephritis—on these appearances, as has been done by some French and German pathologists, appears to us quite unnecessary; since in London, at least, many of the most pronounced granular kidneys are gouty. A certain proportion of the gouty cases are associated with lead poisoning.

Fig. 163.

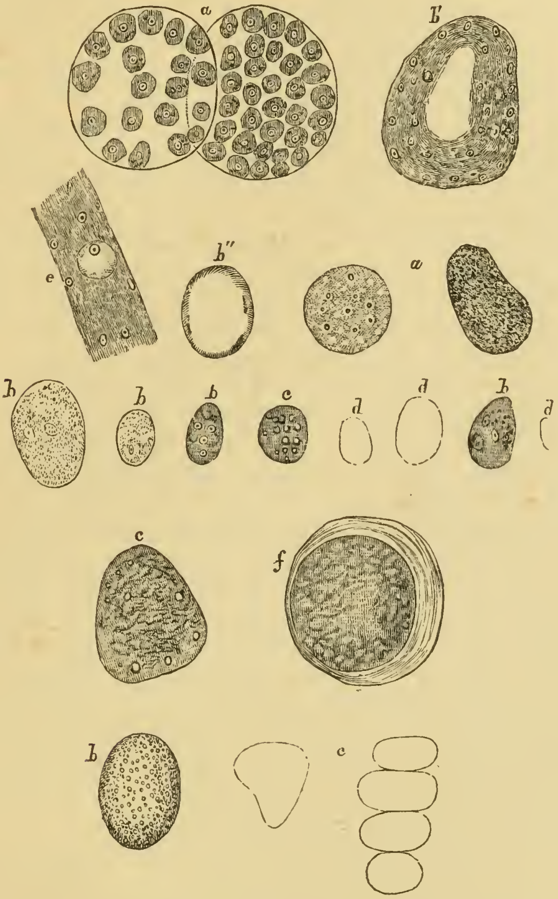


Tufts of acicular crystals, composed of urate of soda from the matrix of a granular kidney.

CYSTS IN THE KIDNEY.

We proceed to the consideration of the cystic growth which often takes place so abundantly in diseased kidneys. These formations appear as vesicles of very various size, ranging from microscopic objects of $\frac{1}{1000}$th inch diameter to the magnitude of a cocoa-nut. They contain usually a clear colourless fluid, which is slightly albuminous, and holds in solution the ordinary salts of the serum.

FIG. 164.



Renal cysts, and cyst-like casts.

a, Cysts containing cells. *b*, *b*, *b*, *b*, Cysts containing granulous matter and nuclei; in *b'* the celloid contents are disposed so as to form an epithelium round a central space; in *b''* the nuclei are elongated. *c*, *c*, Cysts containing granulous and oily matter. *d*, *d*, *d*, Small transparent vesicles. *e*, Young cyst diam. $\frac{1}{2000}$ inch in the remains of a tube. *f*, A cyst with laminated walls. *a*, Two doubtful cysts, probably casts, without distinct envelope, consisting of granulous and a little oily matter. *b*, An oval fibrinous cast advanced in fatty degeneration. *c*, Pale homogeneous fibrinous casts.

Sometimes it is of a dark yellowish colour, and more or less viscid, indicating, perhaps, the presence of colloid matter. We found, in one instance, a large quantity of yellowish granular corpuscles diffused through the fluid, and fragments of an epithelial pavement, consisting of closely apposed nuclei. No urinary principles are found in them, at least as a general rule, but oily matter is not unfrequent, and cholesterine is occasionally present. As they extend and attain a largish size, they seem to cause absorption of the cortical rather than of the medullary substance, so that on laying open a cyst, a medullary cone may be seen at the bottom of it, and forming, perhaps, a ridge in its interior. The whole kidney structure sometimes appears to be destroyed, but the organ acquires an immense apparent size. A case is recorded where the two kidneys weighed 3 lbs. 10 ozs. and 3 lbs. respectively.

Origin of Cysts.—Respecting the origin of cysts, opinions are divided ; Drs. Johnson and Frerichs maintain that they are produced by obstruction of the tubes, and subsequent dilatation from secretion taking place within them. Mr. Simon first proposed the idea that they originate as new formations or growths within the tubes, each cyst having its origin in a germ or nucleus particle, such as under healthy conditions might have produced an epithelial cell. Rokitansky and Paget have also adopted this view, and we have ourselves been long convinced of its accuracy. It applies especially to those cases where the cyst formation is extremely abundant, but we are more inclined to believe that the few and rather large cysts which occur in kidneys not seriously diseased, are produced in the former manner. When the tuft in the interior of a Malpighian capsule is compressed and spoiled, we believe that a cyst may be developed from the capsule in the same way as from a portion of a tube. The smaller cysts contain usually either a clear fluid, or granulous, or an admixture of granulous and oily matter. The larger ones sometimes contain an endogenous cell growth. The envelope in all is well marked, formed of a distinct homogeneous membrane ; it occasionally presents concentric laminæ. Oval and roundish fibrinous casts, when impacted in the medullary tubes, may simulate very nearly the aspect of cysts, especially when some epithelium is imbedded in them.

[To the foregoing account, contained in the first edition of this work, we desire to add the following remarks :—

It seems necessary to draw a distinction between the cysts of considerable though variable size, from a pin's head up to one or two inches in diameter, and those which are quite microscopic and usually found scattered through atrophic kidney substance in immense numbers. In some cases larger cysts are found in kidneys otherwise quite healthy, so as to suggest the idea that they are possibly congenital structures. But in most cases these cysts are associated with contracting granular kidney, and there is great probability in the view which derives them from dilated tubes and Malpighian capsules, the outlet from which is obstructed by fibrous growth. The explanation of the microscopic cysts is more difficult, but according to the views now more generally adopted, they must be regarded as formed either out of portions of tubes shut off during the atrophic process, or out of the contents of the tubes in similar conditions; that is, either from hyaline casts which have assumed a varicose or beaded character, as represented in Fig. 157, or from the same material originally deposited in that form. Dr. Bristowe,* in an elaborate investigation into these structures, has given reasons for both these views, either of which he considers plausible. He has found the microscopic cysts abundant in atrophied kidneys, and comparatively rarely associated with larger cysts. Where the former are abundant, the tubules are always atrophied, and frequently wholly deficient, so that the microscopic cysts and tubes are in inverse proportion to one another. The possible origin of cysts from tubules he explains in the following manner:—When tubules waste, their canals become narrower and narrower, their walls thinner and thinner, till at length they are altogether lost in the surrounding tissue; at the same time they lose their epithelium, and often contain waxy or granular matter. Now, if this obliteration, instead of occurring simultaneously in the whole length, occurred at irregular intervals, producing a moniliform condition of the tubes, and leaving a series of ovoid cavities, there would be cysts, such as those in question; and further, they would be likely to accumulate within them some of the secretions of the tubes, and thus become distended into globular bodies, which might contain all those varieties of contents which are found in diseased tubules. The other explanation, that cysts are formed from *contents* of tubes, Dr. Bristowe regards as supported by the following considerations: (1.) The contents of the cysts resemble those of tubes, being sometimes waxy, sometimes faintly granular, sometimes thickly studded with oil globules, sometimes presenting an epithelial lining, and in one instance containing apparently disintegrated blood. (2.) In cases where casts were present, as well as cysts, linear series of casts were observed within the tubules, some elongated, some ovoid, others globular and, as far as could be seen, identical with cysts. Either view accounts for the presence of cysts in atrophied kidneys and the apparent absence of tubes in the cystic portions.—Ed.]

Urine in Granular Kidney.—There is nothing very characteristic of the urine passed from small atrophied kidneys; it is generally, however, more abundant, sometimes actually in excess, of lower specific gravity, of paler colour, less albuminous, and deposits a less quantity of epithelial sediment than that from the enlarged organ. Fibrinous casts will rarely be entirely absent, but often few in number; they are usually granular and small. Their quantity, and the presence or absence of blood globules, will depend very much on the degree of congestion.

Scarlatinal Nephritis.—The occurrence of renal dropsy after scarlatina is known to be very common, but the precise nature of the renal change is still uncertain. For a long time it has been regarded as the typical form of catarrhal nephritis, or tubal nephritis of Dickinson, and is so described by the majority of authors. Nevertheless, cases occur in which no affection of the tubes can be discovered. In some of these there has been found to be interstitial nephritis.* In other cases all interstitial change is likewise absent, and the observer may be tempted to think there is no structural alteration. A morbid change is, however, sometimes seen, to which the name of *glomerulo-nephritis* has been given; that is to say, a special affection of the Malpighian bodies. The earliest observation on this subject we have found is by Dr. Bristowe,† who, in a case of scarlatinal dropsy in a boy aged sixteen, found, with doubtful changes in the tubes, undoubted changes in the Malpighian bodies, in which some exudation had taken place within the capsule, compressing the tuft of vessels. Klebs describes the whole interspace of the capsule as filled with small, somewhat angular nuclei imbedded in a finely granular mass; while on isolating the glomerulus its surface is found covered with similar nuclei. A singular case was reported by Dr. Wilks, some years ago, of renal dropsy without albuminuria, in which the Malpighian bodies alone were affected; and these were in a state of fatty degeneration, which might very well be a later stage of some such condition as is here described ("Path. Trans." vi. 264). In more than one obscure case of scarlatinal nephritis, where no obvious changes were seen in the tubes or interstitial stroma, we have seen a very large number of nuclei on the glomeruli, but find it difficult to estimate what should be the normal number.‡ It is worthy of notice that the peculiar combination of dropsy with absence of albuminuria, which occurred in some of these cases, is not uncommon in scarlatinal dropsy. This must in any case be distinguished from the transitory parenchymatous degeneration seen in scarlatina, as in other febrile diseases.

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* "Trans. Path. Soc.," vol. ix. p. 309, pl. x.

Distinction of the Two Forms of Bright's Disease.—With regard to the two forms of diseased kidney which we have described, we feel some degree of doubt as to the exact relationship they bear to each other; some regard the atrophied kidney as the more advanced condition of the enlarged one, others consider the two as distinct varieties. We strongly incline to the latter opinion, and to the belief that the enlarged degenerated kidney possesses pathological affinities or analogies with phthisis and the class of scrofulous maladies, while the contracted eminently granular kidney is allied to such changes as those seen in cirrhosis of the liver, and contraction and thickening of the cardiac valves. Between the two forms, of which we have tried to give a typical description, of course there are very numerous intermediate or mixed ones which the student must expect to find.

Combination of different Kidney Diseases.—When the two types of Bright's disease just described are combined, the usual course appears to be for the comparatively acute degeneration (parenchymatous nephritis) to supervene on the more chronic interstitial disease. Lardaceous degeneration is often combined with one of the two diseases just referred to. When combined with parenchymatous disease, it produces the large white form of waxy kidney, in which some portions are opaque with degeneration. But even in this form, and still more in the smaller forms of waxy kidney, there is always a great deal of interstitial nuclear production, constituting a combination with the cirrhotic form of disease. This combination is not easy to explain, since there is certainly no reason to suppose that the lardaceous material becomes organized into fibrous or any other form of tissue, as Dr. Dickinson seems to assume.

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* Cayley, "Trans. Path. Soc." vol. xxi. p. 259, pl. vi. Fig. 3.

† "Trans. Path. Soc." vol. viii. p. 235.

‡ Cases bearing on this subject in Beer, "Die Bindesubstanz der Menschlichen Niere," 1859.

Causes of Bright's Disease.—It is not *à priori* probable that the two forms of disease, being, as we believe them to be, distinct, would be produced by the same causes. The first form, parenchymatous nephritis, is alleged to be produced by exposure to cold, by irritating poisons, and to be the consequence of the acute morbid conditions which arise in exanthematic or other febrile diseases. It is chiefly a disease of early life.

The granular kidney, on the other hand, is closely connected with the chronic processes which lead to fibroid degeneration of various organs, to atheroma of the arteries, and to general senile decay. It is frequently seen in gout, and, in London at least, a certain number of such cases depend upon lead poisoning.

Chronic alcoholism has an undoubted tendency to produce kidney disease, and generally it is thought chronic granular degeneration; but sometimes excessive indulgence sets up an acute morbid process.

We do not think that obstructive diseases of the heart have much to do with the production of renal degeneration, at the most they only act as predisponents. The same may be said of pregnancy, since, though it is, unquestionably, in some instances, by the reflex irritation it sets up, the efficient cause of renal congestion, and consequent convulsions from uræmia; yet Frerichs acknowledges that even among such cases traces of renal degeneration are rarely discoverable. We regard, therefore, albuminuria in pregnancy and in obstructive heart disease only as an indication that the kidney is the seat of an active or passive hyperæmia, which we do not think is often followed by actual degeneration.

Further interesting questions connected with the causation of these diseases our space does not permit us to discuss.

Conditions associated with Bright's Disease.—The granular contracted kidney is often accompanied by changes in other organs, which, though not regarded as its symptoms, are of much importance. These are general tension of the arterial system, thickening of the walls of the smaller arteries throughout the body, atheroma of the larger arteries, hypertrophy of the left ventricle of the heart. Cerebral hæmorrhage* occurs with a certain frequency, and also a peculiar affection of the retina. Some of these changes, especially cardiac hypertrophy, are met with in a certain number of cases of the other form of Bright's disease, but only occasionally; while their absence in the contracted granular kidney is in the highest degree exceptional. Many theories have been framed to explain these morbid conditions, which we can only briefly glance at. Dr. Bright himself noticed the occurrence of hypertrophy of the left side of the heart in cases of granular kidney, and attributed it to the reaction on the heart of obstruction in the systemic circulation; this obstruction being caused as he thought by the fact that blood poisoned, or unhealthy from inactivity of the kidneys, must traverse the capillaries with less ease than healthy blood; as well as by an atheromatous condition of the arteries. Dr. Johnson, who first noticed the condition of the smaller arteries, regards it is as pure muscular hypertrophy, which he attributes to a contraction or "stop-cock" action, intended to hinder or prevent the access of (poisoned) blood to the tissues. The obstruction thus caused to the circulation leads to cardiac hypertrophy. Traube regards the actual obstruction to the passage of blood through the kidney itself as the cause of general high arterial tension, and this as leading to hypertrophy of the left ventricle, notwithstanding the obvious objection that the obstruction (even were it complete) of the renal circulation must have only an inconsiderable effect on the whole mass of the blood. Sir W. Gull and Dr. Sutton have lately put forward an entirely different view of these relations. The kidney disease they regard not as the cause of the other changes, but as itself the consequence of a general arterial disease, to which they have given the name of "Arterio Capillary Fibrosis;' the anatomical characters of which we cannot here consider, but which have led, they think, to the erroneous conception of a true muscular hypertrophy. This change in the vessels, however, causes general obstruction to the arterial circulation, leading in the end to cardiac hypertrophy, and accompanied by arterial tension. The same change in the vessels also gives rise to fibroid change in the viscera, of which the kidney, from its rich vascular supply, is the most conspicuous instance. It is impossible to discuss these views; we can only point out what we believe to be established facts; that a high degree of arterial tension accompanies most forms of Bright's disease, but chiefly the chronic; that true arterial muscular hypertrophy does exist, as described by Dr. Johnson, either as the consequence or as the cause of the arterial tension; and that if the former alternative be accepted, the obstruction must be situated in the tissues; whether or not, for the reason given by Bright. Finally, this condition of tension, if long continued, produces hypertrophy of the left ventricle of the heart.*

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* Mr. Jones speaks of the relation between disease of the kidneys and cerebral hæmorrhage as most intimate, the two lesions concurring in nearly 90 per cent. of his cases. *Vide* "British Medical Journal," 1864, June and December.

While the changes just mentioned do not, as a rule, accompany the parenchymatous, or more acute form of Bright's disease, perhaps on account of its rapid course, there are certain consequences equally associated with either form. These are inflammations of the serous membranes and pneumonia. Dropsy in various forms, as œdema, or effusion into the serous cavities, &c., is constant in the parenchymatous disease, but in the granular form always a late symptom, and sometimes quite absent. Uræmic convulsions and coma often come on in both forms of disease in the last stage. Vomiting is more common in contracted granular kidney. The peculiar affection of the retina referred to above, shown by white patches near the entrance of the optic nerve, and often accompanied by spots of hæmorrhage, is a common and characteristic sign of granular degeneration never seen in the other form of Bright's disease.

NEW GROWTHS IN THE KIDNEY.

Tubercle.—Two forms are known in which tubercle, or scrofulous disease, occurs in the kidney. The first is in the form of discrete miliary granulations, in cases of general tuberculosis; the second more special to the kidney, which may be called renal phthisis.

1. In acute miliary tuberculosis granulations are found in the kidney in a large proportion of cases. The tubercles in acute cases are hard and grey, not more than one-eighth of an inch in diameter. In more chronic cases they increase in size, and become yellowish and opaque—perhaps becoming confluent into larger masses; though it is doubtful where a complete infiltration of large portions of the organ arises in this way. Minute examination shows that the tubercle is formed, in such cases, entirely in the interstitial stroma, which becomes infiltrated over a limited area with the small-celled growth. The tubes in the neighbourhood of the tubercle show mostly a granular degeneration of their epithelium, and become choked with molecular *débris*, but there are sometimes hyperplastic and inflammatory changes, shown by the formation of new cells and hyaline cylinders. We have never seen (in several specimens examined) anything that could be called tubercle within the tubes.

2. In the form which we have called renal phthisis the disease is

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* Bright, "Reports of Medical Cases," 1827. "Guy's Hosp. Reports," vol. i. p. 338, 1836. Bright and Barlow, "Guy's Hospital Reports," 2nd series, vol. i. p. 223, 1843. Johnson, "Diseases of the Kidneys," 1852. "Med.-Chir. Trans.," vol. li. p. 57. Gull and Sutton, "Med.-Chir. Trans.," vol. lv. p. 273. Wilks, "Guy's Hosp. Reports," 2nd ser. vol. viii. p 291, 1853. Traube, "Ueber den Zusammenhang von Herz und Nieren Krankheiten," Berlin, 1856.

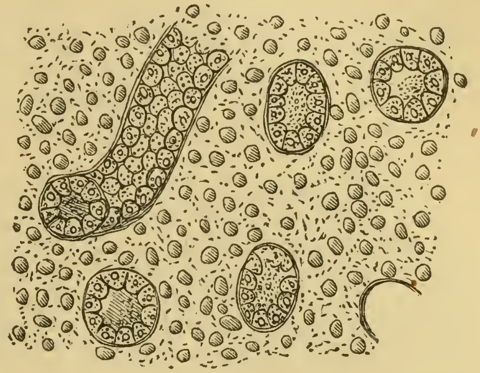
often confined to the urinary organs. The process always begins in the pelvis and calyces, and is sometimes combined with similar disease of the ureters, or bladder. The kidneys are then found either of the normal size or enlarged; sometimes considerably; the surface is usually smooth, but may occasionally show yellow patches or granulations, from the extension of the tubercular disease. On section, larger or smaller portions of the gland, especially near the calyces, are seen transformed into yellow crumbling caseous matter, with some yellow granulations of the same material. Grey miliary tubercles are rarely seen, if ever. The calyces and usually the pelvis also are found covered with an investment of similar soft, yellow, cheesy material, and are dilated sometimes very considerably, and extend at the sides of the cones nearer to the surface, so that the same appearance is produced as is shown in Fig. 167. Sometimes, according to Förster, there is a contraction of these spaces. The next stage is that of softening; cavities opening into the calyces appear to be formed, as in the lungs, by a combination of inflammation with necrosis, and discharge into the urinary passages the so-called "scrofulous pus," which may sometimes be traced in the urine. The process meanwhile extends towards the cortex, till a considerable portion of the organ may be destroyed, and the shape becomes very irregular. Usually both kidneys are affected. The termination of the process is by some pathologists supposed to be such a kidney as represented in Fig. 166, converted into cysts filled with white putty-like matter; but it appears that may be the consequence of simple pyelitis.

With regard to the minute anatomy, Förster states that there is tubercle present, and that it is, as in the miliary form, wholly interstitial. Without being able precisely to confirm or contradict this statement, we must say that the tubes are always seriously implicated, their epithelium granular, and disintegrated; their cavities containing pus cells and hyaline cylinders. It must then be left to future research to determine whether the destruction of the organ is ever due solely to a degenerative scrofulous inflammation, or whether tubercles are always present from the first.

Lymphatic Tumours.—In cases of leuchæmia, tumours of this kind are not unfrequently formed in the kidney as in other organs. They form either distinct whitish soft masses, sometimes projecting on the surface, or else a diffuse infiltration, producing a mottled appearance. The minute structure is represented in Fig. 165. Masses of lymphoid corpuscles are seen interspersed between the tubes, forming an interstitial infiltration, something like the nucleated tissue in granular contracted kidney, but differing in the slightly larger size of the elements and in the entire immunity of the tubes, which look perfectly normal. These masses are not liable to caseation and softening, like tubercle.

In anæmia lymphatica, or cases of adenoid or lymphatic growths without leuchæmia, similar products are seen. Discrete tumours are figured in the paper by Dr. Murchison, before quoted ("Trans. Path. Soc." vol. xx. p. 192, plates vii. and viii.). We have more often seen diffuse infiltrations, which present microscopical appearances precisely resembling those in Fig. 165. Sometimes the capillaries are seen infarcted with white corpuscles, which perhaps accounts for part of the infiltration of the stroma.

Fig. 165.



Section of kidney affected with a lymphatic tumour or lymphatic infiltration. The tubes are seen widely separated by a mass chiefly composed of lymphoid corpuscles. The epithelium of the tubes is unaltered.

Fibroma.—Small fibrous tumours, an eighth of an inch in diameter or more, are not unfrequently seen in the pyramidal portion. They are composed of firm fibrous tissue, and sometimes contain remains of secreting structure, but have no importance. The only case of a large fibrous tumour of the kidney which we have found recorded is that communicated by Dr. Wilks to the Pathological Society, of a man, aged fifty-three, in whom the right kidney was converted into a tumour, the size of a child's head, composed solely of very hard fibrous tissue. ("Trans. Path. Soc." vol. xx. p. 224.) Small lipomata sometimes occur in the cortex. Sarcoma only occurs in secondary growths. A dermoid cyst was observed in one case by Sir J. Paget.

Syphilitic tumours, in the form of gummata, are occasionally seen, but are rare. As in the liver, they may ultimately produce a mere puckering of the organ, closely resembling that produced by fibrinous blocks.

Cancer.—We have not exact data for determining the frequency of renal cancer, but it is certainly not rare. Secondary seems to be more frequent than primary cancer. Contrary to the general rule it occurs equally at all ages, and not only in advanced life, especially if primary cancer only be regarded; but if secondary cancer be included, Dr. Walshe's tables show the period from fifty to seventy to be most liable. Ten cases only occurred in the previous years of life; while nineteen were noted in the succeeding twenty. Scirrhus is rarely, if ever, found; and the same may be said of colloid. Encephaloid growths, especially in children, attain in the kidney an enormous size. A case has been mentioned to us by Dr. T. K. Chambers, in which the weight of the tumour was three-fourths that of the whole body. The statement of M. Rayer, that cancer of the liver and right kidney frequently coexist, as well as cancer of the adjacent parts of the stomach, or descending colon and left kidney, is confirmed by Dr. Walshe. Rokitansky notices that cancer of the kidney often coexists with cancer of the testis on the same side; the renal disease, we think, is most commonly developed after that of the testis. "In thirty-five cases of renal cancer, the disease," Dr. Walshe says, "affected both organs sixteen times, the right alone thirteen times, the left alone six." The urine excreted by cancerous kidneys may long retain its natural characters. When, however, the growth softens and breaks down, blood, puriform matter, or cancerous detritus, may appear in the urine.

Primary cancer in the kidney is usually an infiltrating growth, so that the form of the organ is often preserved. It may begin in the cortex, and gradually involve all but the pyramids; or, on the other hand, begin at the papillæ, within the pelvis, which it dilates, and increases till it is covered only by a shell of kidney substance, sometimes distinctly separable. In such cases it may grow down into the pelvis and ureter. It gives rise, rather frequently, to cancerous thrombosis of the renal vein, and the cancerous thrombus may extend into the vena cava, whence metastasis to the lungs easily results. Infection of the lymphatic glands and neighbouring organs may of course occur, but does not happen so often. Secondary cancer occurs usually in several nodules, and commonly in both kidneys at once.

With regard to the histology of primary renal cancer, it appears to be always true cancer, with alveolar structure and epithelioid cells. According to the most recent investigations it is said to start from the epithelium of the tubes, and to be in the first instance a sort of adenoma, or glandular structure.*

Entozoa occurring in the kidney are the echinococcus and cysticercus. The so-called hydatids from the first of these have, in rare cases, been passed with the urine.

The *adipose tissue*, in which the kidney lies imbedded, may, according to Rokitansky, increase to such a degree as to penetrate by the hilus into the substance of the organ, impede its nutrition, and induce a kind of atrophy. Rokitansky states that, in the highest degree of this change, the kidney presents the appearance of a mere mass of fat, without the slightest traces of renal organization; the urinary passages at the same time being atrophied and obliterated. But in all such cases it is impossible to say that the atrophy of the kidney was not the primary condition, and led to increase of the surrounding tissue.

The *capsule* of the kidney may be inflamed, in consequence of which fibroid thickening may take place, and more or less of induration, atrophy, and obliteration of the organ. The cortical substance is especially apt to be involved, and the surface is sometimes overspread with purulent matter, while the tissue itself becomes sloughy or gangrenous, or is only congested and softened.

Congenital Cysts.—The congenital cystic kidney, different from all the forms of cystic disease before mentioned, must be compared with similar conditions of the ovary and testicle. The whole kidney is found converted into a compound cystic structure, which may sometimes attain so large a size, even in the fœtus, as seriously to interfere with birth. Less extreme cases will of course survive into infantile or even adult life. The condition may go on increasing after birth. A simultaneous cystic degeneration of the liver has been observed.*

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* Klebs, "Path. Anat.," p. 614. Waldeyer, "Virch. Archiv.," xli.

ANOMALOUS CONDITIONS OF THE URINARY PASSAGES.

Under these we comprise the ureters, and their upper terminations, the pelvis and calyces of the kidney. The ureters may terminate, from congenital defect, in a cul-de-sac, either in the vicinity of the kidney or of the bladder. Sometimes they are double or triple, usually from fissure of the pelvis of the kidney, but they generally unite again before their vesical termination.

Dilatation.—It is not uncommon to find them considerably dilated, when the opening into the bladder has been greatly narrowed or obliterated. The calyces expand at the expense of the renal tissue, and extend outwards towards the surface, till at length there remains only a thin layer of the cortical substance compressed against the investing capsule, and the kidney is converted into a number of pouches, separated by membranous loculi, which contain the remains of the medullary cones. The surface of the kidney becomes lobulated in a marked manner from the pouches pressing outward between the interlobular septa. The ureters are at the same time distended, sometimes to that extent that they resemble a portion of small intestine; at the same time their walls are somewhat thickened, so that they do not appear to be much thinner than natural; they only attain, however, a considerable thickness when there is concurrent inflammation. The ureters become also increased in length, and therefore do not lie straight, but are thrown into coils or flexures. Dilated ureters are usually divided by imperfect septa, resembling valves, and it has been supposed that these are new formations; but some indication of such a division is often seen in the normal ureter. Their mucous lining does not appear to be so often inflamed or ulcerates as that of the calyces and pelvis. The pouches formed by the dilatations of these are often filled with puriform fluid, or with a mixture of pus and urine, or even with clear serum only. To the latter condition the term *hydrops renalis*, or *hydronephrosis*, has been given; it is also called sacculated kidney. It seems to take place when the obstruction to the flow of the urine into the bladder is complete, and when in consequence, after extreme distension and atrophy of the renal tissue, the secretion of true urine ceases, and is replaced by a mere serous fluid. After a time, such tumours may diminish, and almost disappear, from the absorption of their contents. The ureters in such cases also contract, and become obliterated. This condition is often called cystic dilatation of the kidney, and must be clearly distinguished from the cystic condition dependent on actual cysts in its substance. It is not necessarily connected with inflammation, as obstruction from any cause will have the same effect. Very considerable dilatation is sometimes produced by stricture of the urethra. In some cases the ureter is

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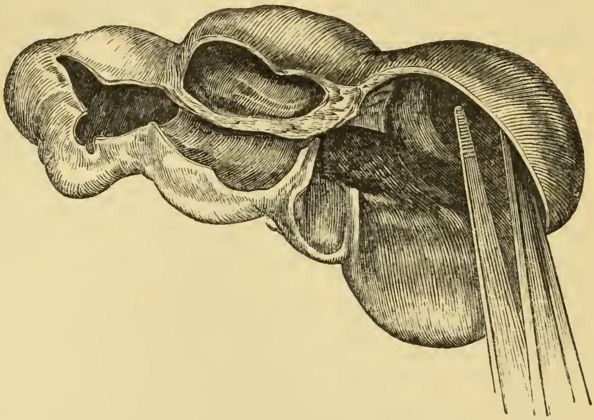
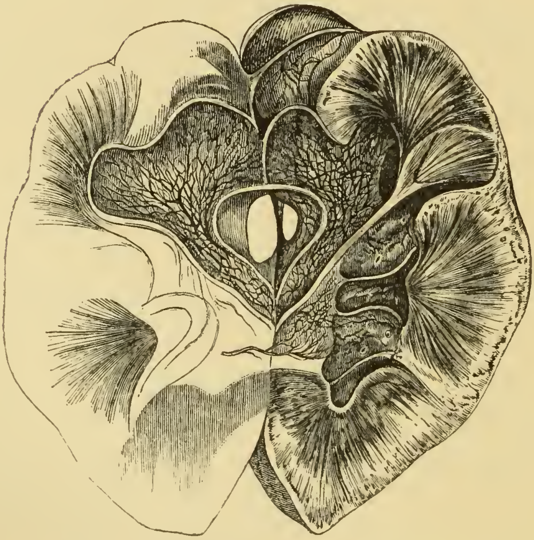
* "Trans. Path. Soc.," vol. vii. p. 229.

Fig. 166.



Cystic dilatation of kidney.

Fig. 1ϵ7.



Pyelitis. There was a concretion in the ureter, consisting of phosphates and animal matter.

blocked just at its entrance into the bladder, without any sign of inflammation of the tube, perhaps from inflammation of the bladder. Injury to the ureter, producing stricture, is an occasional cause. Congenital hydronephrosis, arising from some disease in fœtal life, is sometimes seen; the dilatation may be sufficient to hinder birth, as in the congenital cystic condition.

Pyelitis.—Inflammation of the urinary passages often coexists with, and may be the cause of, the state of dilatation; while obstruction may, on the other hand, lead to inflammation. It is not unfrequently produced by the irritation of calculi, or results from the extension of vesical disease, or from metastasis of this, according to Rokitansky. It appears sometimes to arise idiopathically, or from the general condition of the body. Stricture of the urethra is a common cause of disease of the mucous membrane of the bladder, as well as of dilatation of the ureters. The inflammation set up there is very prone to creep backward, and affect the urinary passages, which are predisposed to it by their unnatural distension, and the prolonged contact of unhealthy urine. The mucous membrane is found in various degrees tumefied, injected, or of a saturated red colour, of villous aspect, and covered with a muco-purulent fluid. Perforation of the ureters may take place in consequence of sloughing, the urine infiltrating into the adjacent tissues, and producing either extensive sloughing, or circumscribed abscesses. The inflamed mucous membrane, in many cases, causes phosphates, or a mixture of these and carbonates, to be deposited from the urine. When atrophy of the kidney takes place, these saline deposits, cemented together by mucus, form a "yellowish-white, greasy, and chalky pulp, or putty-like material, which fills the calyces," and is enclosed by the wasted organ as by a cyst. This form of destruction of the kidney, and the pyelitis from which it arises, are often regarded as due to tubercular disease, and then we hear of "strumous" or "scrofulous" pyelitis. But it certainly may arise from ordinary inflammation; and when the disease is confined to one kidney, or when no tubercular disease is present elsewhere in the body, it almost certainly is so. It does not seem to occur unless the ureter be obstructed. In other cases, a renal calculus, of the kind described by Dr. Prout as the Phosphatic, may be produced in this way. A clot of blood is sometimes thought to be the nucleus. Uric acid calculi are rather the cause than the result of inflammation.

Croupous or Diphtheritic Pyelitis.—Rokitansky mentions the occurrence of inflammation, which he distinguishes by the name Exudative, from the above catarrhal form, as a secondary affection in cases of serious blood disease. It is observed in typhus, in the exanthemata, in diphtheritis, and acute tuberculosis, and also in pyæmia, and occasions the formation of unhealthy fibrinous effusions upon the mucous surface, associated in some cases with hæmorrhage.

Cysts, containing a glutinous or hard (colloid ?) matter, about the size of millet seeds or peas. are occasionally found developed under the mucous membrane of the urinary passages.

Tubercle.—Rokitansky states that this "is always a symptom of tubercular disease, that has spread from the male genitals to the urinary organs." We think some cases recorded in the "Transactions of the Pathological Society," and some that we have seen ourselves, show that this is by no means necessarily the case. It is most frequent in the ureters when the kidneys are involved at the same time, but we have seen it in them when the kidneys were healthy. Usually there exists at the same time tuberculosis of some important organ, as of the lungs or the hip joint. The deposit takes place in the submucous tissue, and forms, when its progress is chronic, grey granulations, which become yellow, soften, "and give rise to small circular ulcers." When the disease is more acute, larger patches of deposit are formed, or "the mucous membrane becomes infiltrated throughout with the tubercular product of inflammation, which is at once detached as a cheesy, purulent mass."

Cancer but rarely attacks the urinary passages, and hardly ever, except it is elsewhere in process of development. The disease may extend to them from the kidney, from the lumbar glands, or from the bladder, either when it is itself primarily affected, or involved in uterine cancer.

Cases of villous tumours, perhaps cancer, and certainly primary, of the pelvis of the kidney, resembling villous disease of the bladder, have been recorded. ("Path. Trans.," xxi. 239 and 241.)

Parasites.—*Strongylus gigas* (p. 213) inhabits the pelvis of the kidney, or stretches into the ureter, and causes wasting of the kidney. *Distoma hæmatobium* (p. 214) inhabits the renal vessels, and its eggs are discharged by ulceration into the urinary passages, producing hæmaturia and albuminuria. Stricture of the ureters, and hydronephrosis may also result, and even calculus.

ANOMALOUS CONDITIONS OF THE BLADDER.

This receptacle is subject to various *congenital malformations*, o which we shall only mention a few. The first is termed inversion or prolapsus of the bladder, and results from a defect in the lower part of the abdominal parietes, with corresponding defect of the anterior wall of the genito-urinary canal. There appears in the hypogastrium "a red, mucous, dilated prominence, the edges of which join with the common integument; in the male sex it passes downwards, so as to terminate in the fissure of the urethra ; in the female it is surrounded by two diverging tumours, which represent the labia, and it terminates in the lamina of the general integument which invests the rima vulvæ." The symphysis pubis is wanting; a vestige only of the umbilicus is sometimes seen at the upper part of the tumour. The openings of the ureters are seen at the lower and lateral parts of this mucous surface, which is, of course, the posterior wall of the bladder. In a case which we saw recently, the penis was very short, and the canal of the urethra open above in its whole length. In less severe cases the abdominal wall only is deficient, the bladder being perfectly closed, and appearing as a red tumour—*Ectopia vesicæ.* Slighter degrees of the same defect occur. The second malformation is attended with fissure of the opposite side of the bladder, and of the adjacent cavities, so that a kind of cloaca is formed, similar to that which exists in the lower animals. In some rare cases the urachus remains pervious. so that when urine is passed it escapes at the umbilicus. In others, again, the bladder has no external opening, the communication with the urethra is not formed.

Dilatation of the bladder is no uncommon occurrence, and may be occasioned either by paralysis of the muscular tunic, or by some obstacle to the outflow of the urine, as stricture of the urethra. We think that the amount of dilatation is greatest when the muscular coat is paralyzed, and that in the other class of cases, where some obstruction exists, the great hypertrophy of the muscular fibres which is induced prevents the distension becoming so great. Rokitansky, however, seems to consider obstruction as the most powerful cause of dilatation of the bladder. The effect of the stronger contractile coat of the bladder in preventing dilatation is shown in some cases recorded in the Transactions of the Pathological Society, in which it is mentioned that the ureters were much distended, while the bladder was contracted, or not dilated. The bladder may be so dilated as to rise above the umbilicus considerably, and to contain twenty pints of urine. The paralytic dilatation depends, we believe, in some cases, on fatty degeneration of the muscular coat.

This dilatation may be congenital, and so extreme as to be an obstacle to birth. Diverticula, or partial dilatations of the bladder, are not infrequent. They are always found in cases in which the muscular tunic is hypertrophied, and seem to be produced by protrusions of the mucous membrane taking place between the fasciculi, which are subsequently pushed outwards more and more by the pressure of the urine. The lateral portions, the posterior surface, or the neighbourhood of the fundus, are the situations in which diverticula usually form. They have no muscular tunic, except, occasionally, a few scattered fibres, which Rokitansky suggests may be some evidence of their being congenital. Calculi get into these pouches sometimes, and become so lodged and concealed as to escape detection by the sound.

Contraction of the bladder most often is rather apparent than real, and depends on irritation of the mucous lining, with hypertrophy of the muscular coat. Sometimes it is partial, or may cause a kind of hour-glass constriction of the cavity. When a calculus is present, the walls are sometimes found closely embracing it; and a case is mentioned by Morgagni, in which the bladder was so closely contracted around a needle, that there was scarce room for anything more in its cavity.

Hypertrophy of the muscular coat is observed in cases where that tissue is unusually exercised, and is often of a manifestly beneficial tendency. When the mucous lining is irritated by the contact of unhealthy urine, perhaps in some degree inflamed or ulcerated, the reflected stimulus from the spinal cord becomes more intense, and the contractions of the muscular fibres more energetic. Again, when in consequence of stricture, the difficulty of expelling the urine becomes great, the contractile force is increased to meet it, and this increase is occasioned by the greater exertions which are necessary. It is sometimes accompanied by dilatation, sometimes there is no enlargement or even diminution of size. One of the commonest causes is obstruction caused by enlargement of the prostate. The appearance of the inner surface of a bladder whose muscular coat is hypertrophied, is peculiar, and is well compared by Rokitansky to that of the right ventricle of the heart. The muscular fasciculi become unusually prominent, and by their divisions and interlacements produce a kind of irregular network into the meshes of which the mucous membrane dips, and through which it may be forced in sacculi. The technical term for this condition is *columnated*, or in the French original, "vessie à colonnes." Dr. Walshe has seen polypoid growths from the inner surface of the bladder, consisting of prolongations of the mucous and submucous tissue, in a state of simple hypertrophy. If we except these, and the diverticula before mentioned, it does not appear that there is any true hypertrophy of the mucous lining. Rokitansky has, in rare cases, seen the mucous membrane *atrophied*, "reduced to a very delicate, shining membrane, resembling the arachnoid," while the muscular coat almost entirely disappears.

The bladder is liable to various *displacements*. It may form the contents of inguinal, vaginal, and perineal herniæ, it may be introverted and forced into the urethra, and even in females project from the meatus urinarius externally.* When hernia of the bladder occurs, it is either in part, or, more rarely, completely destitute of peritoneal covering. This depends on the anterior part of the viscus, which has no serous covering, being the first to prolapse; but, as the organ descends, the posterior part which is lined, carries with it the peritoneum, and thus forms a sac, into which intestine or omentum is often protruded.

Hyperæmia of the bladder of a passive kind occurs when there is some obstruction to the free passage of blood through the pelvic veins and the vena cava; it is, therefore, associated generally with a similar condition of the adjacent viscera. It produces increased secretion of mucus, sometimes spots of extravasation, and occasionally such dilatations of the veins as have been termed vesical hæmorrhoids. These, however, do not appear like the common little tumours of the rectum, but rather as prominent and distended vessels.

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* In the Report of the Pathological Society, 1852-53, there is an instance recorded by Mr. Pilcher, in which about two-thirds of the bladder were extruded from the abdomen through the inguinal canal, and lodged in the scrotum. The hernial portion was large enough to contain 50 oz. of fluid.

Inflammation of the bladder is much more often seen in a chronic than in an acute form; this depends partly on the acute stage of the disease, in recent cases having generally subsided before death occurs, partly on the greater frequency of cystitis which is chronic from the commencement. The appearances in *acute cystitis* are strong vascular injection of the mucous lining, with brownish patches in the vicinity of the neck and fundus; more or less thickening of the membrane, with exudation of fibrine or pus on the surface, or foci of the latter in its substance. The mucous tissue may be ulcerated at several points, softened, or affected by commencing gangrene. Abscesses may form in the substance of the parietes, and open either into the cavity of the bladder, or upon its external surface. Sometimes the mucous membrane is almost completely destroyed, a few shreds or filaments being the only traces remaining, while the muscular tunic is left as if cleanly dissected. This is probably the result of phagedænic ulceration.

Chronic cystitis may be the condition resulting from one or more attacks of the acute form, or may be produced by the extension of urethral inflammation, or by the irritation of unhealthy urine, or of calculi. Its characters are various degrees of vascular injection, mingled with dark-reddish, slaty, or bluish-black discoloration, more or less tumefaction of the mucous membrane, with secretion of mucus, or muco-pus, often in considerable quantity. Sometimes, from the irritation excited, the muscular coat becomes hypertrophied and columnated; but the more ordinary condition in chronic cystitis, is the thickening and more or less uniform induration of the parietes, which assume an homogeneous, lardaceous appearance, doubtless from their infiltration with exudation matter. It not unfrequently happens that an acute attack, or exacerbation, supervenes upon a state of chronic inflammation. The following abridged account of the appearances which then present themselves is taken from Rokitansky: "The bladder is found dilated, and filled with decomposed, intensely alkaline urine, mixed up with blood of a brown colour, viscid mucus and pus, sanies, lymph, and detached portions of mucous tissue, in the shape of discoloured flocculi, or larger patches." The mucous membrane, incrusted by a deposit of amorphous and crystalline phosphates, is sometimes "of a dark-red colour, appears spongy, softened, and pultaceous, is easily detached and bleeds; when chocolate-coloured or greenish, it is found purulent, infiltrated with sanious matter, or converted into a friable, flocculent tissue, which is traversed by the urinary sediment." In some cases, the submucous and muscular tunics are exposed, and are in various stages of softening, suppuration, and disintegration. As the morbid action advances outwards, the peritoneum at last may become involved, and general inflammation of this membrane may be set up. Ulceration, attended or not with suppuration, sometimes extends deeply and gradually, and at last perforates the walls, when extravasation of urine takes place, if not prevented by inflammatory exudation, and adhesion of adjacent parts. The further progress of ulceration sometimes forms a communication with the cavities of the adherent viscera; in this way the walls of the rectum, the colon, and the ileum, have been perforated, and their contents have made their way into the cavity of the bladder.

Chronic or subacute inflammation of the bladder is very commonly an attendant upon paraplegia, and proves the immediate cause of death. The inflammation is set up, we conceive, in the same way as that of the eye is when the fifth pair of nerves has been divided, and results from loss of the nutrient power of the tissues, and consequent stagnation of blood in toneless vessels. At the same time, the urine rendered alkaline by the decomposing influence of the vesical mucus upon the urea, reacts, no doubt, upon the inflamed membrane as a further cause of irritation. The urine is turbid with quantities of muco-pus and detached epithelium, contains often albumen, sometimes blood, and always prisms of the triple phosphate. The urine under these circumstances always contains bacteria or vibriones, like those seen in decomposing urine out of the body. It has been thought that these must be introduced into the bladder from without by the process of catheterization, which is often required in such cases. Nevertheless, bacteria have been seen in the urine when it was quite certain no catheter had been used. The coats of the bladder undergo similar changes to those above mentioned, but of a marked asthenic character. The mucous membrane is congested, and thickened and altered by fibrinous exudation, or purulent, or sanious; it is incrusted by a phosphatic deposit, and in parts may be gangrenous. The muscular coat is also more or less affected, and the submucous tissue.

Croupous or Diphtheritic Cystitis.—Rokitansky describes exudative processes of a croupy kind, as not very unfrequent in the bladder; they occur in the course of exanthematic diseases, in pyæmia and typhus. The exudation does not affect generally a large surface, but is limited "to round spots or striæ." The mucous membrane beneath the exudation is more or less injected, tumefied, and indurated, or in processes of lower character is softened and converted into a pulpy, gelatinous, sanious, or purulent mass, or even becomes gangrenous.

Some false membranes discharged from the bladder have no connection with croupous or diphtheritic processes, but seem to be due to simple exfoliation of the mucous membrane; which appears to occur from inflammation, or from simple retention of urine, as for instance in the puerperal state. The cast consists of the whole epithelial covering, incrusted with urinary salts. In women it has been discharged during life, and perfect recovery has ensued.

Rokitansky mentions what we have once observed ourselves, an eruption of minute miliary vesicles, containing a clear serosity upon the surface; they accompany, he says, catarrhal inflammation and slight exudative processes, as well as Asiatic cholera. Acute and chronic inflammation of the muscular coat of the bladder are both spoken and written of, but the former appears to take place only as a part of general cystitis, and the latter, if it intend more than hypertrophy of the muscular fasciculi, is only that general infiltration of the parietes, with induration matter, which we have before noticed.

Pericystitis, however, seems to be a more distinct affection; it consists in the spontaneous inflammation of the cellular tissue surrounding the bladder, arising either as a primary or a secondary process. It is to be regarded, Rokitansky says, as a localization of pyæmia. From its original seat, it is apt to spread to the areolar tissue round the rectum, to the anus, and into the scrotum; it may involve also the coats of the bladder, and cause perforation of them. It is sometimes of a chronic form, and then gives rise to induration, rigidity, and callosity of the bladder.

Softening of the mucous membrane, not resulting from inflammation, was observed by M. Louis only twice out of five hundred autopsies; in these the tissue was converted into a kind of pale mucilage. Rokitansky has seen it only once, in a case of typhus.

Tubercle is infrequent in the bladder, and it usually arises by secondary extension from the sexual organs in the male, less frequently by extension from the kidneys. It has never been observed in the female sex, nor is tubercle formed here in cases of general tuberculosis. It is only met with in the form of separate granulations, which are surrounded by more or less hyperæmia, according to the rapidity of their production; these soften and give rise to circular ulcers of the mucous membrane covering them. The cervix and fundus are the parts chiefly affected.

Cancer is much more often seen in the bladder as the extension of disease from contiguous parts, than as the primary phenomenon. This at least seems to be the more general opinion; but Dr. Walshe affirms that primary vesical cancer is far from being so uncommon as is generally supposed, and we are quite inclined to agree with him. Scirrhus is very rare in the vesical parietes. Mr. Coulson has never seen it, nor has Sir B. Brodie, except where it constituted part only of a morbid growth. Rokitansky mentions having seen it extending over large surfaces of the sides of the bladder. Encephaloid forms nodules in the walls of the bladder, which lead, on the one hand, to ulceration, on the other hand, to projecting growths on the surface. Adhesion to the neighbouring organs, perforation and fistulous openings into the rectum, vagina, ileum, or peritoneum, may result.

Villous cancer, or cauliflower-like excrescence, is the form which vesical cancer commonly assumes. This may be of very various consistence, and often very vascular, easily bleeding, and situated especially at the trigone, the neck, the fundus, and the vicinity of the urethral orifices. It is developed in the submucous tissue, but as it grows, the mucous membrane is destroyed, and either an ulcer is produced, or a soft, luxuriant, fungous mass. It produces irritation of the bladder, more or less difficulty in micturition, and, in the latter stages, hæmorrhage, which may be considerable, and difficult to arrest. The urine contains mucus, sometimes blood, cancerous detritus, and portions of encephaloid matter at various times when fragments happen to become detached.

Simple papillary outgrowths are also confounded under the same name of cauliflower excrescence, and their distinction from cancer is very difficult. They are composed of simple fibrous tissue, covered with thick layers of vesical epithelium which, from its great variety of form may easily, when it appears in the urine, be confounded with cells of cancer. Hæmorrhage and other symptoms like those of villous cancer occur. It rarely happens that, as in a case reported by Dr. Dickinson, masses of tissue are expelled during life which can be positively recognized as cancer ("Trans. Path. Soc.," vol. xx. p. 233).

MORBID CONDITIONS OF THE URETHRA.

Congenital Malformations.—We notice the following malformations:—Fissure on the upper surface (epispadias), or on the lower (hypospadias); the former, when extending the whole length, occurs as a complication of eversion of the bladder; the latter accompanies fissures of the scrotum, and occasions a resemblance to the female conformation. The urethra may terminate at various points of its normal course, in the perinæum, the root of the scrotum, or anywhere between this and the glans; the opening in these unnatural situations is very small, and sometimes is completely closed (atresia urethræ). A kind of cloacal formation may also be produced by the urethra terminating in the rectum, or in the female in the vagina. The diameter of the canal may be congenitally narrowed at the extremity, or at other parts, and dilatation of the bladder, or cystic dilatation of the kidneys may result.

Contraction, however, is much more commonly the result of inflammatory disease, under which head we shall describe it.

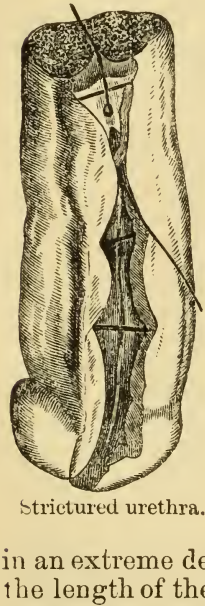
Dilatation of the urethra is often produced by some obstruction to the flow of urine; it occurs, for the most part, in the membranous portion, which is expanded into a pouch, sometimes as large as a small orange. The mucous lining of these pouches is usually "injected and thickened, presenting fungous vegetations, and occasionally coated with lymph." Dilatation may also be caused by a calculus. The urethra is frequently *distorted* from its normal direction, either by the dragging of a large scrotal herniæ or hydroceles, or by the pressure of tumours. Enlargement of the lateral lobes of the prostate pushes it to one side, of the middle lobe divides it into two passages. The length of the canal in such cases is increased.

Lacerations of the urethra may be produced by mechanical injuries, by the passage of fragments of calculi, or by ulcerative destruction. They often give rise to urinary fistulæ.

Urethritis; Gonorrhœa.—Inflammation of the urethra of the catarrhal kind is exceedingly common, and constitutes the misnamed gonorrhœa. It commences at the anterior extremity and gradually proceeds backwards, in very severe cases extending even to the bladder. The mucous lining becomes swollen, injected, and covered with mucous or muco-purulent secretion. Its follicles and lacunæ are attacked, especially the lacuna magna; in the chronic state they are enlarged and relaxed, and pour out as well as the general surface a thin mucous, so-called gleety, discharge. During the acute stage, when the inflammation extends deeper to the fibrous structure of the corpus spongiosum, exudation of fibrine sometimes takes place in the venous sinuses, which renders them incapable of distension, and thus occasions during erection a bending of the penis towards the affected part which is termed *chordee.* Abscess may form also in the same situation from suppuration of the exuded fibrine, or, perhaps, also from severe inflammation of the lacunæ. The inflammation may spread along the continuous mucous lining to other adjacent parts, to Cowper's gland, the prostate, the vesiculæ seminales, and the testicles. This extension of the morbid action to other parts is commonly attended by a subsidence of it in its original seat, so that it is often questionable whether actual metastasis has not occurred. The gonorrhœal discharge passes through the same stages as that from other inflamed mucous surfaces; it is at first a thin mucous fluid, then more tenacious and muco-purulent or purulent, and as the inflammation subsides, it becomes again thin and pale. When a chancre coexists with gonorrhœa, "the discharge has usually a greyish or reddish tint, or sanious aspect."

The mucous follicles in the vicinity of the meatus are liable to be specially affected both in the male and female; Dr. Oldham appears to refer to this condition under the name of follicular inflammation of the vulva, and Kleeberg, as quoted by Dr. Adams, thus speaks of their condition in the male: "The orifices of the lacunæ become closed by inflammation, and in the course of two or three days pustules are formed in their places, which break and discharge a yellow pus. The orifices of the large mucous follicles are now seen dilated and surrounded by a swollen dark-red border, and they discharge a muco-purulent fluid into the urethra." The disease sometimes assumes a chronic form. The contact of unhealthy vaginal secretion is the most common cause of urethritis, and it is important to be aware that this effect may be produced by the fluids of females who are perfectly chaste. The great acidity of the normal vaginal moisture may be a cause in some instances. Stimulating injections, the irritation of calculous fragments, the presence of stimulating diuretics in the blood, the materies morbi of gout, of influenza, and the suppression of cutaneous eruptions, are mentioned as causes of this inflammation. Abrasions and excoriations of the urethral mucous lining are occasionally found when it is inflamed, but ulcerations are (probably) always the effect of other causes. The syphilitic poison producing urethral chancre, the presence of foreign bodies, calculi, &c., the irritation caused by a stricture in the part behind, softening tubercle, are so many causes of more or less extensive ulceration. It is said by Mr. Adams to occur in rare cases spontaneously.

Fig. 163.



Strictured urethra.

Stricture of the Urethra.—A very frequent result of inflammation is stricture. This consists in a narrowing of the canal from some organic change in the structure of the part itself, or in that of those around. It may affect any part, but is most frequent at the junction of the spongy and the membranous portion. Out of 189 cases examined by Mr. Phillips, the seat of the stricture was in 138 from four to six and a half inches distant from the meatus. Contusions and wounds occasion stricture of urethra, which in severe cases of the latter is extremely intractable. The simplest form of stricture is when the canal is partially occluded by a fold of membrane passing across it; this may be of such a shape that a crescentic or sometimes an annular opening is left. Several of these strictures may coexist in the same urethra ; as many as eight are said to have been observed by Calot. It is thought that these result from the healing of an ulcer, or the raising up of a fold of the lining membrane. In the more common kind of stricture (which occurs in the spongy portion) the urethra is narrowed in a much greater extent of its course, and sometimes in an extreme degree. Half an inch or an inch is not uncommonly the length of the contracted part, and sometimes the whole extent of the spongy portion is affected. The stricture occupies sometimes one side, at others it completely encircles the canal. It is not difficult to understand the mode of its production, which is very illustrative of the general contractile tendency of exudation matter. Effusion and inflammatory new growth take place during inflammation in the mucous tissue itself, or in the submucous, as well as sometimes in the corpus spongiosum ; if this be not absorbed, it passes into the state of fibroid or induration matter, and continually tends to shrink up and contract into a narrower space. In proportion as this takes place the canal must be contracted. The mucous membrane lining the indurated part is often ulcerated and destroyed, commonly from the mechanical effect of catheters pushed against it; but it may also take place spontaneously, and it has happened that the indurated part being destroyed by the extending ulceration, the stricture has thereby been cured. A more common and less favourable result of deep ulcerations is the perforation of the canal and the formation of a fistulous opening. When the obstruction occasioned by a stricture is very great, and it may be such that the passage will hardly admit a bristle, the urethra behind is dilated, often inflamed, and sometimes ulcerated, so as to give rise to urinary fistula, or effusion of urine. The bladder and ureters are affected, as we have before described. Of course the hypertrophy of the bladder, by propelling the urine more forcibly against the stricture, must tend to increase the dilatation of the canal behind.

The urethra is sometimes obstructed by warty growths, which are situated generally near the meatus, and are remarkably vascular; they are developed as the result of gonorrhœa, and polypous growth is occasionally found, but is much more rare. Chronic disease of the lacunæ sometimes converts them into small indurated tumours, which become imbedded in the corpus spongiosum; of this kind, perhaps, is an instance mentioned by Rokitansky, in which numerous cartilaginous protuberances from the size of a millet seed to that of a pea were scattered over the surface as far back as the bulb, not, however, obstructing the passage. It may be well to remind the student that the common expression of "old cartilaginous strictures" intend simply the density and firmness of the induration matter, and not at all that it contains any true cartilage. Stricture is rare before puberty, but has been found at the age of ten years; it is well to be aware that it may at this age be possibly the result of the habit of masturbation. Many surgeons have regarded the excessive use of stimulating injections as an occasional cause of stricture.

Croupous, or *fibrinous* inflammation, in very rare cases occurs primarily on the urethral mucous lining, and chiefly in children. It is probable that it takes place also here as a secondary process in the same diseases in which it is found in the bladder and ureters. Variolous pustules are not unfrequent in the urethra. *Tubercle* is of rare occurrence in the urethra; it is only present when the entire urinary apparatus is likewise affected. It has been found in the miliary as well as in the more massive form. *Cancer* affects the urethra, either in the male or female, for the most part as an extension of adjacent disease; but cases are recorded where the growth in this part was either primary or isolated.

Morbid Conditions of the Female Urethra.—Displacements of the uterus, especially retroversion, cause compression of the passage, as also does the pressure of the child's head during labour. Such compression not unfrequently produces sloughing of the parietes and vesico-vaginal fistula. *Dilatation* of the urethra is in rare cases congenital; it is sometimes effected purposely for the sake of removing calculi from the bladder, and may be safely carried to the extent of permitting a stone one inch and a half in diameter to be extracted. Paralysis, however, has sometimes resulted, and consequent incontinence of urine from excessive dilatation. Prolapsus of the bladder alters the *direction* of the canal of the urethra, so that it passes upwards and forwards. There is no essential difference between catarrhal inflammation of the female and of the male urethra ; it is generally consecutive to a similar condition of the vagina. The lips of the meatus are seen to be swollen, and on pressure upwards muco-pus flows from the orifice. Stricture is very rarely, indeed, the result of inflammation, which appears to be owing chiefly to the circumstance that the disease in the female is not of long duration. Mr. Curling met with a case in which a stricture, attended with complete retention of urine, was produced by contusion experienced during a severe labour.

The *vascular tumour* of the meatus is thus described by Sir Charles Clarke : "Its texture is seldom firm ; it is of a florid scarlet colour, resembling arterial blood ; and if violence is offered to it, blood of the same colour is effused. It is exquisitely tender to the touch ; and if an accurate examination of it be made, it appears to shoot from the inside of the urethra. Its attachment is so slight that it appears like a detached body lying upon the parts." Sometimes the growths extend partially along the urethra, or may even be situated at the neck of the bladder. The tissues of the urethra occasionally undergo a kind of chronic hypertrophy, so as to form "a bulbous tumour." The veins are enlarged and varicose, and the areolar tissue increased in quantity, while the mucous membrane may be either thick, or on the contrary, thin and shining. A mucous discharge takes place from the canal and from the vagina. We are much inclined to consider this state as more truly deserving the name of chronic urethritis than that which Dr. Ashwell has so denominated, but which seems more of the nature of severe pruritus. It may also be compared to the state of the veins of the rectum, which gives rise to hæmorrhoids.

DEPOSITS AND CONCRETIONS IN THE URINARY TRACT.

Various chemical substances are deposited from the urine under certain morbid conditions in different parts of the urinary tract. We only notice here those which form masses visible to the naked eye.

Deposits in the Uriniferous Tubes.—Uric acid and urates, chiefly urate of ammonia in a crystalline form, are sometimes found precipitated in the straight tubes of the medullary portion of the kidney in new-born infants. The papillæ and pyramids appear to the naked eye marked with yellow or orange lines, and sometimes the same yellow sandy material lies in the pelvis. Microscopic examination shows the straight tubes completely infarcted with the material. This deposition appears to take place after birth, especially in the first fourteen days ; and has never been found in a child actually born dead, very seldom in those who live but one day ; hence it may, in some cases, be valuable as evidence that a child has actually lived. This singular condition is found in about half the children who die between the second and fourteenth day ; but is evidently transitory, and may therefore be more frequent than this proportion suggests.

Similar infarctions of urates are seen occasionally in the kidneys of gouty persons, and perhaps precede the deposition of crystalline urates in the stroma, which has been already described.

Calcareous infarctions are sometimes seen in the straight tubes. They are filled with granular carbonate of lime, which also encrusts the papillæ. This has been seen in cases where bony parts have been rapidly absorbed, as in mollities ossium.

Concretions in the Urinary Passages and Bladder.—The concretions or calculi which form in the pelvis of the kidney and in the bladder have so much similarity that they can be described together. Probably many calculi are first formed in the kidney, and find their way into the bladder, where they increase in size. The process by which they are formed is not a simple precipitation, since they always have a basis of animal matter which doubtless modifies the form in which the mineral matters are arranged. This animal basis appears to be some modification of albumen, or perhaps mucin.

Calculi vary in size from the finest sand to that of a child's head. The very large stones are now exceedingly rare. Some foreign body, or a blood-clot, or mass of mucus or pus, sometimes forms the nucleus. Among the largest known are one in the museum of St. Thomas's Hospital, weighing twenty-five ounces, and one at Cambridge, weighing thirty-two ounces.

The following are the chief classes of calculi : *—

1. *Uric acid* calculi are very common. Their shape is round or oval, surface smooth, or nearly so, structure composed of concentric laminæ. If pure uric acid they are white, but are usually coloured yellow or pink. The size varies from the finest sand to as much as twenty-five ounces (that mentioned above).

Urate of ammonia composes calculi very similar to those of uric

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[*] Perhaps the most successful attempt yet made to explain the mechanism of the production of calculi is that of Dr. Vandyke Carter, who has investigated their microscopic structure, and finds that the forms in which the mineral salts are precipitated, coincide perfectly with those in which (as shown by Mr. Rainey) similar salts are deposited in the shells of animals, and which have been reproduced artificially by the last-named investigator by mixing saline solutions with organic matters. The process termed by Mr. Rainey, its discoverer, molecular coalescence, we believe to be the key to the formation of calculi, but can only here refer to the original memoirs bearing on the subject, viz., Rainey, " On the Mode of Formation of Shells, &c.," London, 1858; V. Carter, " On the Microscopic Structure of Urinary Calculi," London, 1873 ; see also Ord, " Quarterly Journal of Microscopical Science," vol xii. new series, p. 219, and Harting, Ibid. p. 119, " On the Artificial Production of Organic Calcareous Formations."

acid; the other salts of uric acid are mostly found in mixed calculi.

2. *Oxalate of lime* forms what is called the mulberry-calculus, from its rough tuberculated surface. It is extremely hard, laminated, and stained dark-red or brown from altered blood. Colourless crystalline calculi of the same composition are sometimes found in the pelvis of the kidney; and small polished grains, like poppy-seeds, have been found to be chemically the same. The two forms last-mentioned occur in the kidney. Oxalate calculi are very common.

3. *Phosphatic* calculi are mostly composed of phosphate of ammonia and magnesia, or triple phosphate, mixed with some phosphate of lime (called the fusible calculus). They are smooth, white, round, or oval, generally laminated, and earthy, but sometimes made up of white scales. Calculi of pure phosphate of lime are rare, but resemble in appearance those just described, except that the colour is usually pale brown. Both these forms may occur in the kidney as well as in the bladder.

4. *Cystin* calculi are rare. They are yellowish-white, smooth, generally uniformly round or oval, but when occurring in the kidney may be irregular in shape. The substance is made up of shining crystalline plates. They may occur in the kidney, but more commonly in the bladder.

5. *Xanthin*, discovered as composing a calculus by Dr. Marcet, in 1817, is of extraordinary rarity. Only two subsequent specimens have been recorded, one by Langenbeck, one by Mr. Taylor, from the museum of the College of Surgeons. ("Trans. Path. Soc.," vol. xix. p. 275.)

6. *Carbonate of lime* forms by itself very rare calculi, but is not uncommonly found in mixed calculi.

Calculi of pure carbonate of lime are brownish or white on the surface, the substance pure white, and either granular or semi-crystalline and translucent. In one instance they have been found in the kidney.

7. *Mixed calculi* are probably the commonest of all. They consist of a nucleus of one material surrounded by a coating of another, or sometimes of alternating layers of two substances. These are chiefly uric acid, or urates and triple phosphate; but oxalate and carbonate of lime may both form part of mixed calculi, the latter as an external crust. Mixed calculi occur more especially in the bladder, but sometimes in the kidney. The deposit of phosphates on the outside is connected with an inflammation of the bladder and alkaline urine.

CHAPTER XXXIX.

ABNORMAL CONDITIONS OF THE MALE GENERATIVE ORGANS.

Testicles and Vasa Deferentia.—There is no sufficient evidence to show that more than two testicles ever exist. They are absent when the entire sexual apparatus is wanting, and in some rare cases they are imperfectly formed, or one only may exist. An apparent absence of one or both glands at birth is not very unfrequent, the descent of the organ being arrested or delayed, so that it lies in the groin, the inguinal canal, or the lower part of the abdomen. It may happen that the vas deferens exists without any trace of a properly formed testicle. If this be the case on both sides, virile power may be apparently unimpaired, though there is, of course, perfect sterility. Again, the vas deferens may be absent, and even the epididymis, while the testicle is plump and well formed, and though incapable of fulfilling its function, scarcely less than the other. The most usual and least degree of imperfection is, that the vas deferens terminates in a blind extremity before reaching the vesicula seminalis.

Retained Testis.—A defect of greater practical importance as being more common, is the imperfect descent of the testis into the scrotum. Of one hundred and three male infants examined by Wrisberg at the time of birth, seventy-three had both testicles in the scrotum, while in twenty-one one or both were in the groin, and the remainder had one or both in the abdomen. He found the imperfection more frequently on the left than on the right side, in the proportion of seven to six. Mr. Curling believes that if the descent does not take place within twelve months after birth, it is seldom fully and perfectly completed afterwards without being accompanied by hernia. The reason of this is sufficiently apparent, the pressure of the muscular walls of the abdomen must tend to cause the descent of the intestine through the open inguinal canal. When the testicle is still in the abdomen at birth, it may descend, and usually does, within a few weeks (it did so in ten out of the twelve cases mentioned by Wrisberg), or it may not descend till some time before puberty, or it may not appear at all. The cause of the testis remaining in the abdomen is considered by Mr. Curling with much probability to be owing either to paralysis and defective development of the cremaster muscle, or to the contraction of adhesions between the gland and some adjacent viscus. The discovery of the continuation of muscular fibres from the fixed attachment of the cremaster up along the gubernaculum to the testis in its primitive situation by the side of the vertebral column inclines us strongly to believe that these fibres must be the agents in causing the descent of the gland into its appointed place. Contraction of the external abdominal ring is also mentioned as one of the causes impeding the descent of the testis. In rare instances the testis wanders into other situations—one has been found in the perinæum, the other being normally placed; and in a few instances a testicle has preferred to make its exit by the crural instead of the inguinal canal. Sometimes it happens that the gland is turned round in the scrotum, so that its anterior face becomes posterior.

What is the condition of the gland in these cases of imperfect descent? This must always be a matter of much anxiety to the subject of the phenomenon, and it is not easy to answer the question with certainty. Mr. Curling, indeed, affirms, as the result of long-continued and most careful investigations into the subject, that "spermatozoa have not been discovered after death in the spermatic ways of a detained testicle in any one instance that he knows of."* Of course, where only one testis is so situated it is of little consequence, the remaining healthy organ preventing its owner from being sterile, but where both are undescended, the many cases in which these testes have been found atrophied or completely undeveloped, render it impossible to speak with certainty of their condition, although instances are not wanting of the subjects of this deficiency becoming fathers of healthy children.†

It is probable that an imperfectly descended testicle is more prone to certain diseases than is the healthy gland. There are on record not a few cases of malignant disease affecting a retained testis—a condition attributed by Virchow partly to the increased liability to injury in this position, and partly to the predisposing developmental imperfection.

There is no such condition known as true *hypertrophy* of the testes, although the size and weight of healthy testes vary considerably in different individuals.

Atrophy of the testis, either congenital from defective development or acquired, is not unfrequent. Several instances are mentioned by Mr. Curling, in which the penis and testicles of persons arrived at the age of puberty, or of adults, did not exceed the size of those of children; two of these were of weak mind, but this condition is by no means the frequent accompaniment of cretinism or idiocy. A case recorded by Mr. Wilson shows the influence of aroused mental emotions in producing the due development of the generative organs, which had not taken place at the twenty-sixth year of age. The atrophy of the testis in old age comes on very gradually, the organ becomes flabby, and its tissues discoloured, but it is seldom diminished in size.

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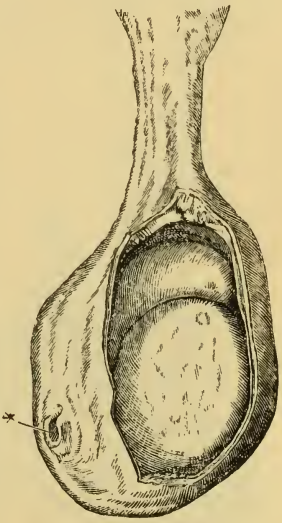
* "A Practical Treatise on the Diseases of the Testis, &c." by T. B. Curling, F.R.S., third edition, 1866, p. 30.

† See a case in "Trans. Path. Soc.," vol. xix. p. 288, in which a married man, aged sixty, having two testicles no larger than filberts, but otherwise healthy-looking, had six children. No spermatozoa were found, but the testes did not seem atrophied, only undeveloped.

Mr. Curling states that the ordinary weight of a sound testicle, in a healthy adult, is about six drachms, great individual differences, however, being often met with, as well as differences between the two glands; the left was heavier than the right in five cases out of six. If the weight fall below three drachms, the organ may be certainly said to be in a state of atrophy. In one instance known to us both testes were reduced to the size of small peas, and the individual, aged twenty-three, presented all the characteristics of a eunuch. "A testicle in an advanced state of wasting, not arising from disease of the gland, usually preserves its shape, but feels soft, having lost its plumpness, elasticity, and firmness. The tunica albuginea is thin. Its texture is pale, and exhibits few blood-vessels; the tubuli and septa dividing the lobes are indistinct, and the former cannot be so readily drawn out into shreds as before. The epididymis does not usually waste so soon, nor in the same degree, as the body of the testicle. It sometimes, however, loses its characteristic appearance; and I have even found it reduced to a few fibrous threads. The fluid pressed out of the wasted testicle and epididymis is entirely destitute of spermatic granules and spermatozoa. In many instances adipose tissue is deposited behind the tunica vaginalis, and encroaches on the epididymis, and the posterior part of the testicle. Fatty matter is also found in the glandular substance. The structures composing the spermatic cord undergo a corresponding diminution, the cremaster muscle disappears, the nerves shrink, and the vessels are reduced in size and number. The vas deferens, though small, can generally be injected with mercury as far as the commencement of the epididymis, and sometimes the metal reaches the vasa efferentia."*

FIG. 169.



Inflammation of tunica vaginalis, after application of caustic, the aperture made by this is shown at *. There are flocculi of lymph on the serous surface of the testis. (From Mr. Curling's article.)

The causes of atrophy of the testis are very various: deprivation of its supply of blood from obliteration of the spermatic artery, injuries of the spinal cord, producing paraplegia, and probably therewith the loss of the nervous influence necessary for the gland, inflammation of its tissue—especially syphilitic inflammation—varicocele, over-excitement of the organ, the long-continued use of iodine, which is said to affect the female breast similarly, tubercular elephantiasis, injuries to the back of the head and nape of the neck, the pressure of effusions, and of large herniæ, neuralgia, and any wasting disease,—all appear, on good evidence, to be occasional causes of atrophy of the part in question.

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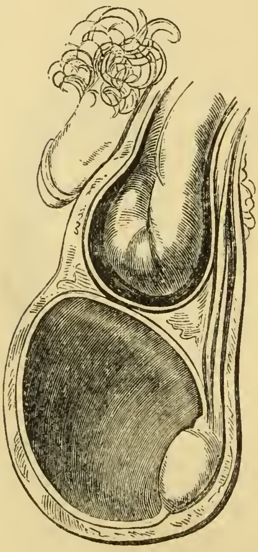
* Mr. Curling, *Op. cit.*, p. 65.

The serous covering of the testis, the tunica vaginalis, is liable to be attacked by *acute inflammation*, either primarily or by extension from an inflamed testicle, and then suffers as other serous membranes do. The membrane becomes thickened and injected with blood, and is coated with a more or less considerable quantity of fibrinous exudation. Serum is at the same time effused into the cavity, and is rendered turbid by flakes of fibrine floating in it; and in rare instances suppuration of the membrane takes place. The unabsorbed fibrine very commonly forms adhesions between the opposing surfaces, which, in time, become very firm and dense, and may obliterate the cavity.

Hydrocele of the tunica vaginalis is the name given to the pear-shaped, translucent swelling caused by an undue accumulation of the fluid which is secreted by the serous membrane forming the tunica vaginalis. This accumulation may depend upon either an excessive secretion or a defective absorption of this fluid, and it is usually caused by some inflammatory action. The fluid is usually clear, and of a straw colour, but it may be quite colourless, green, turbid, slightly ruddy, or glistening with shining scales of cholesterine. Its quantity is sometimes very considerable; six quarts are said to have been withdrawn in the case of Gibbon, the great historian. The serous membrane in old hydroceles may be more or less thickened, and even the seat of calcareous deposit. Adhesions formed between the two layers of the tunica vaginalis may, according to their length and extent, alter the usual position of the testicle, so that it appears in front, instead of lying in its usual place at the posterior and lower part of the distended sac; or they may subdivide the cavity, and produce thus a multilocular hydrocele. The natural cul-de-sac which exists between the epididymis and the body of the testicle, is some-

Fig. 170.



Drawing of large hydrocele, combined with scrotal hernia.

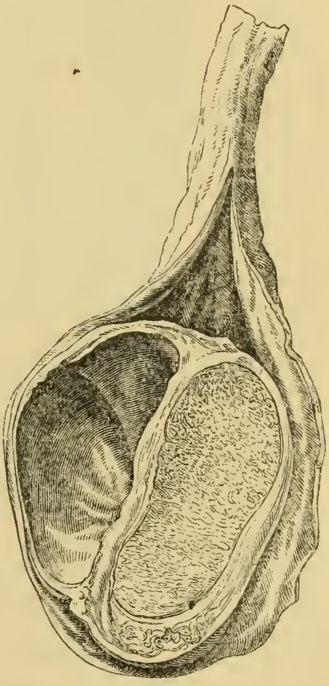
Curling's article on "Testis" Cyclop. Anat. and Phys.

times much distended, so as to form a pouch, which projects on the inner side of the gland. The morbid action in hydrocele is confined to the serous membrane; the testis either remains natural, or is somewhat flattened, and, in some cases, partially atrophied by the pressure of the fluid. When, however, the original seat of disease is in the gland itself, the serous covering is often secondarily involved, so that serous effusion in the sac is very often associated with chronic orchitis, or other diseases of the testicle; and such a combination has been termed a *hydro-sarcocele.* Hydrocele seems to be met with on either side in about an equal proportion of cases.

In *congenital hydrocele* the dropsical tunica vaginalis retains its fœtal communication with the peritoneal cavity.

Encysted hydrocele of the testis, as it is called, proceeds from the development of new cysts beneath the serous membrane. These are exactly similar to the simple cysts we have described, p. 156, having a wall of thin fibrous tissue, and a lining of tesselated epithelium, with usually limpid fluid contents. They may be situated "(1) beneath the visceral portion of the tunica vaginalis, investing the epididymis; (2) between the testicular portion of the tunica vaginalis and the tunica albuginea, which are thus separated from each other; (3) between the layers of the loose or reflected portion of the tunica vaginalis." In the two last mentioned situations they rarely occur. When formed on the epididymis, they sometimes carry the serous membrane outwards as they enlarge, so that they become pedunculated, just as the small serous cysts in the neighbourhood of the ovary often do with the peritoneum. Mr. Curling states that these pedunculated cysts do not acquire a large size, seldom exceeding that of a currant. Several cysts may coexist in the same gland, and, when opened, produce the appearance of a sacculated arrangement. The walls of the cysts are liable to inflammation, which causes their contents to be mingled with various exudations of serum, fibrine, or even blood. Spermatozoa are very frequently present in the fluid of encysted hydroceles, to which they impart a milky or opaline opacity. They subside to the bottom of the vessel, where

FIG. 171.



Encysted hydrocele of tunica vaginalis. (From Mr. Curling's article.)

The cyst is between the tunica albuginea and the tunica vaginalis of the testis.

the fluid is left at rest, leaving the upper portion more transparent, but containing some albumen, which is not found in the limpid contents of ordinary cysts of this kind. It is extremely probable that they make their way into the cysts in consequence of rupture of some seminal canal lying in contact with them, just as a biliary duct sometimes opens into the sac of an hydatid. This opinion is confirmed by the circumstance, that patients generally report the swelling to have commenced after some injury to the testicle. Where this is not the case, it has been suggested that the cyst originates in the persistent embryonic tissue, forming the corpus innominatum of Giraldès, which lies on the back of the testis, between it and the epididymis. Its structure is tubular, and it is connected with the ducts of the epididymis, so that the explanation of the passage of spermatozoa into a cyst formed in this position is sufficiently easy.*

Diffused hydrocele of the spermatic cord is the name given to an accumulation of fluid in that portion of the serous pouch carried down with the testicle from the peritoneum, which extends from the internal abdominal ring to the upper part of the tunica vaginalis. This tube may be either wholly or partially obliterated, and it is when it is closed only at the internal ring above, and at the testicle below, that an effusion of fluid into it gives rise to this form of the affection.

Encysted hydrocele of the spermatic cord forms a tumour, of oval shape, and seldom attaining the size of a hen's egg. It is loosely attached to the vessels of the cord which pass behind it. Instead of there being only one cyst, there may be several, forming a series along the cord. This circumstance indicates their origin, from the partially obliterated process of peritoneum, which is carried down with the testicle in the formation of the tunica vaginalis. According to another view, they are simple cysts, of new production, analogous to those which constitute encysted hydrocele of the testis. It may be mentioned, that simple hydrocele occurs not unfrequently together with some of the other varieties, and also with inguinal hernia.

Hœmatocele is the term applied to a tumour formed by an effusion of blood from the vessels of the testis or its coverings, or of the spermatic cord. Its most common seat is in the tunica vaginalis, which may be so greatly distended as to exceed the size of the adult head. The blood undergoes various changes, coagula being formed sometimes in separate masses, sometimes in firm layers, as in the walls of an aneurism. The fluid part in old cases is more or less thick and grumous, sometimes resembling coffee-grounds. The presence of the blood occasionally excites inflammation, leading to fibrinous and serous effusion, and, it may be, to suppuration. The effused blood sometimes putrefies, offensive gases are produced, and, unless free exit be given to the decomposed matter, fatal gangrene takes place. The tissues surrounding the tunica vaginalis are apt to become involved in the inflammation, though they are affected in a more chronic manner, and thus the walls of the serous cavity are considerably thickened—perhaps so as to be half an inch in diameter. The testicle usually remains unaffected, except that in old cases it is atrophied from pressure. An encysted hydrocele of the testicle or epididymis may be converted into an hæmatocele, by the effusion of blood into its cavity. So, also, may the encysted hydrocele of the cord. Both these, however, are rare affections.*

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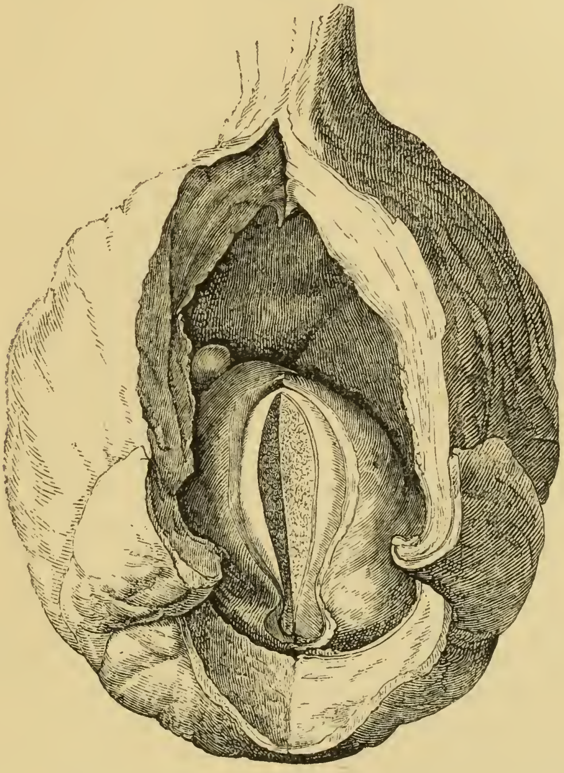
* See a note on this subject by Mr. J. W. Hulke, F.R.S., in the "Trans. Path. Soc." for 1870, p. 277.

Fig. 172.



Hæmatocele; tunica vaginalis greatly thickened, testis pretty healthy. (From Mr. Curling's article.)

Diffused hæmatocele of the cord results from the rupture of some vessels of the cord, which are, probably, in some way diseased (*e. g.* varicose), in consequence of which blood is effused, in greater or less quantity, within the spermatic fascia; and if the bleeding continues, or recurs after an arrest of varying length of time, a tumour of enormous size may be formed, reaching down even to the knees, as in a case recorded by Mr. Bowman in the Medico-Chirurgical Transactions, vol. xxxiii. The cause of the rupture is generally some straining effort.

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* See a case recorded by Mr. Henry Arnott, in the "Trans. Path. Soc.," vol. xxii. p. 184, of old hæmorrhage into the cellular tissue of the scrotum, simulating cancer.

Orchitis (ὄρχις, a testicle)—inflammation of the testis—may be either acute or chronic. The acute disease is sometimes primary, as when the testis has suffered from external violence; but the more common condition, in which the testicle swells with great pain and tenderness during the course of urethritis, is really an epididymitis, the inflammation extending from the affected part along the vas deferens. This limitation may be usually easily perceived, the swollen epididymis far exceeding the body of the testis in size, although still retaining its characteristic form.

Opportunities rarely occur of examining the gland when acutely inflamed, but the following appearances have been observed in cases of secondary inflammation of the organ: the testis itself is not much altered, but the epididymis is enlarged, especially at the lower part, to twice or three times its natural size, and feels thick, firm, and indurated. This enlargement depends on the presence of exudation matter in the intertubular connective tissue. The coats of the vas deferens are thickened, and the adjacent vessels injected. The tunica vaginalis is inflamed, and its walls glued together by soft adhesions, or separated by a variable amount of fluid. Suppuration rarely occurs in the body of the testis in consecutive orchitis, it is more frequent in primary; indeed, in the former, it is not uncommon for the gland itself to escape entirely. When pus has been formed in the testis, it does not easily make its way out, and consequently burrows in different directions, disorganizing the tissue of the gland. Sometimes, when all active inflammation has subsided, the fluid part of the pus becomes absorbed, leaving only an encysted cheesy mass behind. Another result of inflammation of the testis is wasting of the gland, occasioned by the interstitial fibrinous exudation passing into fibroid tissue, and compressing the blood-vessels and tubuli. The enlargement of the epididymis not unfrequently subsides very incompletely, leaving an indurated, knotty swelling, situated usually at its lower part. The fibrinous matter which imbeds the duct and the areolar tissue in this part, does not necessarily cause the obliteration of its canal, which is even sometimes considerably dilated, as Mr. Curling has observed, so as to be four or five times its usual dimensions. At the same time it must be borne in mind that the prolonged continuance of this fibroid induration of the tail of the epididymis may, in certain cases, lead to permanent obstruction of the excretory duct.

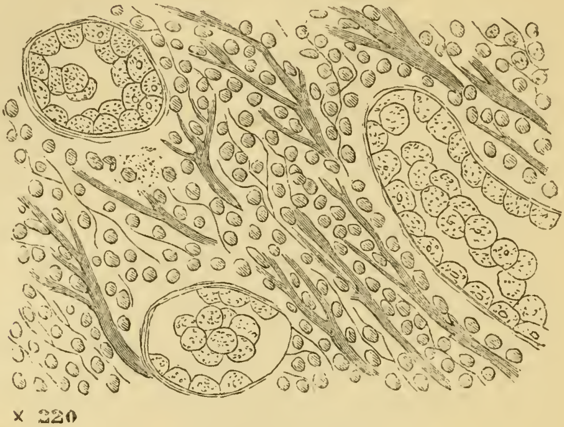
Chronic orchitis, in which the inflammatory changes already described proceed more slowly and in somewhat different fashion, rarely develops spontaneously. It may indeed follow a blow, or take the place of the acute affection; but in almost all cases the change may be traced to the presence of a syphilitic taint, or to the tendency to chronic inflammatory affections which characterizes that constitutional state which is commonly styled the "scrofulous or strumous diathesis." The nature of these varieties, as well in their anatomical changes as in the different treatment required for their relief, render it necessary to discuss them singly. It requires some care to distinguish simple chronic inflammation, or syphilitic disease, from scrofulous, or so-called tubercular testis. Hence the name which any particular specimen receives is very often dependent merely upon history or accompanying morbid changes in other organs. The form of disease which we shall describe as "scrofulous orchitis" is by some authors regarded as the type of chronic inflammation, by others as really tubercular. But from the former it is sufficiently distinguished by its essentially degenerative or destructive character; and, as to the latter point, tubercles need not be present at all, unless we take this word in a very wide sense; while the disease in which tubercles are present from the first shows very different features. The scrofulous form might very well be called "phthisis of the testicle"; and in regard to the question of ætiology, it is important to remember that this is one of the conditions which may be the antecedent of general miliary tuberculosis—a fact which we have observed in more than one well-marked instance.

Syphilitic orchitis, or *syphilitic sarcocele*, is the chronic inflammation of the testicle met with in the later stages of constitutional syphilis. It consists mainly of a smooth, scarcely tender, general enlargement of the body of the testicle. Very generally both testes are affected, one following soon after the other. There is seldom any pain, save some aching in the loin, caused by the dragging of the heavy organ, nor is there much tenderness. In the earliest stage of the disease a distinct nodulation of the surface may be made out, but as the disease progresses the nodules become fused together in a general smooth indurated enlargement of the organ. The skin is implicated only in exceptional cases, and the epididymis is as rarely involved in the disease—points of considerable diagnostic importance, in the absence of other symptoms of constitutional mischief, as scaly eruptions, nodes, or affections of the throat.

The exciting cause of the enlargement is obscure; in certain cases it seems due to excessive sexual excitement, but it is hardly ever met with in connection with urethritis.

The anatomical changes present are of two kinds, either a diffuse form of cell proliferation in the intertubular connective tissue, commencing at the surface and dipping in towards the centre of the organ, or the formation in the body of the testis of gummy nodules, which subsequently become fused together. In the early stages one sees the delicate connective tissue to be crowded with spherical corpuscles, resembling leucocytes, separated by a scanty amount of homogeneous material; but as the disease progresses the corpuscles fattily degenerate, whilst the intervening substance increases in density, and presents the appearance of a tough fibroid network. These degenerative changes commence in the central portions of the nodules, the resulting tough yellow masses being surrounded by a vascular zone of actively proliferating tissues. As the affection subsides, this vascular zone, by an increase of its fibroid over its corpuscular elements, forms a tough capsule to the now cheesy encysted mass.

Fig. 173.

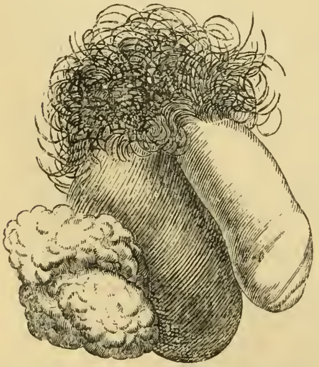


Syphilitic Orchitis.

(From a drawing by Mr. Arnott.)

Meanwhile, however, the changes in the connective tissue, by pressing upon the tubules of the testicle, cause a thickening of their coats and a fatty degeneration of their lining epithelium, so that in advanced cases of the disease the secreting power is gravely impaired, and the physiological activity of the organ diminished, or wholly destroyed. Indeed in some instances, after all active inflammatory changes have subsided, the testicle gradually dwindles away to a hard fibroid mass, not larger than a hazel nut, and of course destitute of functional power. This form of diseased testicle is apt to be associated with hydrocele.

Fig. 174.

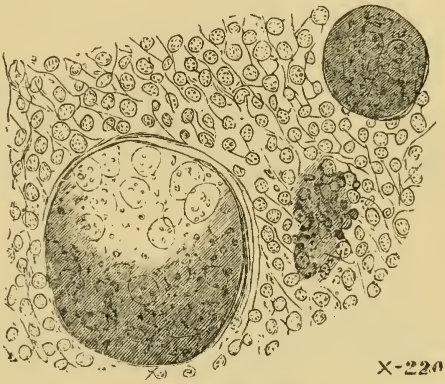


Chronic orchitis, with fungous protrusion of testis.
(From Mr. Curling's article.)

Scrofulous orchitis, although here also similar changes in the connective tissue and the tunics are met with, presents some striking contrasts to the syphilitic form of the disease. The epididymis is now the main seat of the destructive process, although the body of the testicle is frequently involved. The enlargement—often nodular at the outset, and limited to the epididymis—progresses even more slowly than in syphilis, and it is common for the skin to share in the inflammation, the coverings of the testis becoming early adherent to it; and finally, the skin breaking down, an ulcer is formed, having thick indurated edges, through which the pent-up diseased testis protrudes in a granulating mass, in the form of a "benign fungus." This fungous protrusion, although it may be covered with ordinary granulations, includes a great portion—sometimes even the whole of the testicle—the altered tubules being separated by a certain amount of fibro-corpuscular or cheesy material.

The researches of Mr. Curling and Dr. Andrew Clark long ago pointed out the nature of the change chiefly present in this scrofulous form of orchitis. This seems to consist essentially in an affection of the true glandular structures, rather than of the tissues binding these together, although both suffer to some extent. The epithelium lining the seminal tubules proliferates, the cells block the tubes, and dilate them irregularly; nutrition and physiological function being interfered with, the cells become cloudy and swollen, fat granules accumulate within them, they burst, discharging granular nuclei, and finally a considerable mass of oily and fibrinous *débris* occupies the seat of the tubes which have been destroyed. Where these changes have occurred in isolated bits of the tubes, minute grey or yellow nodules result, and hence from their resemblance to so-called "tubercles" in the lungs of phthisical people, these testicles have been called "tubercular." The close analogy between "catarrhal pneumonic phthisis"—in which the main feature is an active proliferation and degeneration of the epithelium—and scrofulous orchitis, on the one hand, and between "chronic interstitial pneumonia," or "fibroid phthisis" and syphilitic orchitis on the other, may serve to render the important distinctions here referred to more evident.

Fig. 175.



Scrofulous orchitis.
(From a drawing by Mr. Arnott.)

Tubercle of the testis, in the form of grey miliary semi-translucent points, such as are met with in the lungs and other organs of patients dying with acute tuberculosis, seems to be a comparatively rare affection. It is probable, however, that this is because the testicles are seldom examined in medical autopsies. In the Museum of St. Thomas's Hospital there is a beautiful specimen of this disease, taken from the body of a man, the subject of general tuberculosis, and showing the testis somewhat enlarged, and thickly beset with grey firm semi-translucent pin-head nodules, none broken down, nor the seat of any notable fatty change.

These miliary granules are usually seated in the delicate intertubular connective tissue, and tend, by their coalescence and subsequent degeneration, to form irregular iron-grey, or partly cheesy masses, indistinguishable in their later stages from the nodules already described under the head of scrofulous and syphilitic orchitis.

Indeed, in all these inflammatory affections of the testicle, although up to a certain point the pathological changes may be tolerably distinct, a stage is reached before long in which all the tissues of the organ partaking in the destructive processes, the tough grey or yellow nodule resulting affords no histological clue to the mode in which the changes have been brought about. For an inflammatory process inside the tubes will speedily set up irritative changes in the surrounding connective tissue, whilst this connective tissue will not long continue inflamed without involving to a greater or less extent the epithelial elements of the tubes adjoining.

Secondary abscesses have been found in the testicle, apparently as the result of pyæmia, in connection with synovitis of the shoulder joint and lobular pneumonia. A preparation of this kind is in the Museum of St. George's Hospital.

TUMOURS AND NEW GROWTHS.

Cancer is met with occasionally in the testis, occurring there almost invariably as a primary disease, the most frequent form being soft carcinoma, called by Abernethy "Medullary Sarcoma." Some part of the body of the testis being first affected, the new growth gradually invades the whole organ, entirely replacing the normal tissues, and involving to some extent the tunics, but rarely infiltrating the skin, save in exceptional instances of tumours fungating through an opening in the scrotum. The fibroid alveolar stroma so characteristic of this form of cancer is usually well marked, but the cell forms contained in its meshes are sometimes so small and regular as to suggest rather a lymphoma or small round-cell sarcoma than true carcinoma. In one such case which we examined, the histological appearances of the testicle-tumour were very peculiar, the structural arrangement being quite that of carcinoma, but the cells being all spherical—not much larger than leucocytes—and contained in long parallel slit-like meshes of the stroma. In this instance, however, the patient died some months later, and in large secondary formations in the lumbar glands, and scattered through the abdomen, the ordinary elements of carcinoma with large epithelioid cells were abundantly present.

Even where the mass of the growth is composed of large irregular cells packed in a slight fibroid framework, there are usually portions—generally about the margin—of the tumour in which the cells are small and regular, like those of granulation-tissue.

True scirrhous carcinoma, in which the dense stroma is greatly in excess of the contained cell growth, is hardly ever encountered in the testis, although different parts of the same tumour often present very different degrees of hardness.

If any of the healthy gland-tissue is left it is spread over and closely adherent to the carcinomatous nodule. This disease has an unhappy tendency to spread up the cord—sometimes as a uniform and continuous enlargement, at others in a nodular, irregular manner—and it early affects the lymphatic glands lying in front of the lumbar spine; the inguinal glands remaining free unless the scrotum also be involved.

Secondary formations in remote viscera are also to be looked for in these cases, if the patient live long enough. The spermatic vessels become enlarged in proportion to the extent and rapidity of the growth.

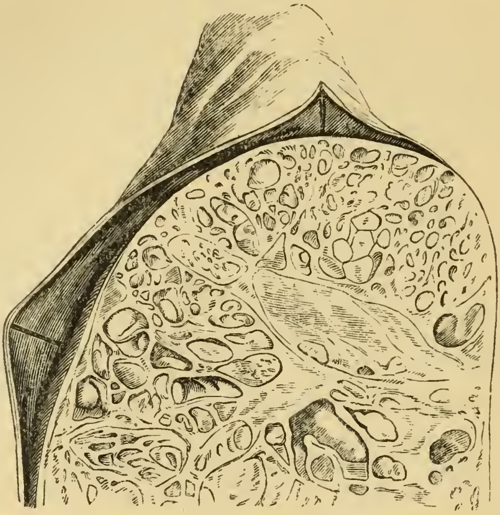
Carcinoma of the testis seems to be most frequent in middle life, and from thence on to sixty or sixty-five, but it has been observed even in infancy.

Colloid carcinoma is unknown in the testis, and there are very few cases on record of *melanotic* cancer in this locality.

A *small round-cell sarcoma* has been described as causing a swelling of the testicle, very difficult to be distinguished from carcinoma. Probably the microscope alone could decide between them, for this round-cell tumour is frequently associated with invasion of the lymphatic glands. It forms a cream-like infiltration of the testis, with an abundant milky juice, and having a structure of round or oval nucleated cells imbedded in a granular semi-fluid matrix, but with none of the alveolar fibroid stroma characteristic of carcinoma.

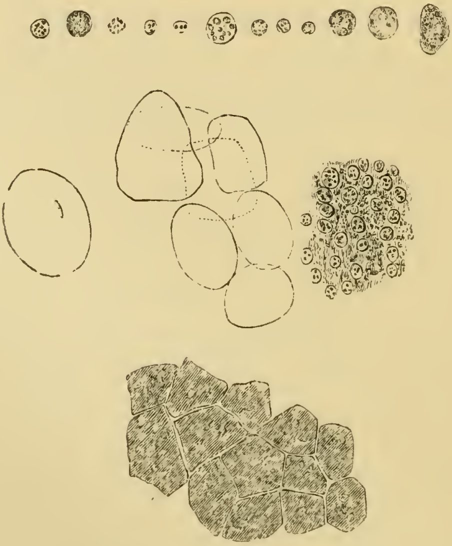
Spindle-cell sarcoma, having a strong tendency to be associated with cyst development, is also met with in the testis. This cystic tumour, like cancer, is limited to one testicle, but differs materially from it in its comparative freedom from the clinical features of malignancy. The cysts may be only two or three in number, or excessively numerous, causing considerable enlargement. They vary in size, from the most minute to the dimensions of a pigeon's egg, and their delicate walls are lined with a beautiful epithelium. Their contents are, in the younger cysts, a transparent, light-coloured fluid, in the older, a more thick, viscid, and very albuminous substance, in which more or less blood may be present. The cysts are imbedded in a stroma of spindle-cell tissue; sometimes small masses of enchondroma being developed between them. A lobulated growth of the same fleshy tissue sometimes arises from the wall of the cyst, and occupies its cavity more or less completely. In one specimen we have examined, the contents of the cysts were not identical; in some, which appeared as opaque white spots, they consisted of layers of scaly epithelial particles, more or less flattened and pressed together; in others, the contents were a pulpy mass of swollen

FIG. 176.



Section of cystic sarcoma of the testis. After Mr. Curling.

FIG. 177.



Contents of various cysts in the case referred to in the text.
In the lowest figure the epithelial scales are flat and apposed to each other; in the middle the epithelial scales are much swollen, and mingled with granular matter and corpuscles.

scales, with very abundant amorphous and oily matter, and small, delicate vesicular globules; others again contained a clear fluid, and a soft, whitish pulp, consisting almost entirely of small oil-laden, granulous vesicles. The stroma enclosing the cyst cavities consisted of a dense fibroid substance. Hæmorrhage may take place into a number of the cysts at different places, as is well seen in a beautiful specimen of the disease in St. George's Museum. In this case the testis was enclosed in a common covering—probably the tunica albuginea—along with the tumour, and lay at one side of it; in other cases, the glandular structure was expanded over the growth. There was no contamination of the glands, nor, as far as known, any return of the disease, in the patient from whom the tumour just mentioned was removed.

In those rare cases in which the appearance of a cystic sarcoma of the testicle has been followed by secondary growths in the lungs and elsewhere, these visceral growths are generally made up of the same spindle-cell tissue as that forming the fleshy stroma of the primary cystic tumour, and are rarely themselves cystic. Such tumours have been commonly described as mixed cancerous and simple cystic growths in the testicle.

Enchondroma.—It has been mentioned that small masses of enchondroma are occasionally developed between the cysts. The tendency to the development of cartilage—usually of a fœtal, hyaline type, but with smaller and more crowded cells—in most new formations in the testicle is singularly marked, the testicle resembling in this respect the parotid gland. This admixture of cartilage with the other connective-tissue elements of the sarcomatous tumours is also commonly to be traced, although in a less degree, in the secondary growths in the viscera and lymphatic glands in cases of diffusion of the disease. The cysts are supposed to take their origin generally in a dilatation of the seminiferous tubules, but they probably also arise from a softening of the sarcoma tissue, or collection of fluid between its histological elements, as seen in other cystic growths elsewhere. They never contain spermatozoa, and the lining epithelium does not resemble that lining the seminal tubules; indeed, in one reported case* the cysts were lined with ciliated epithelium. We have seen a *cholesteatoma*, or pearly epidermic tumour, in the epididymis attached to the testicle.

Osseous growths are unknown in the testicle, the nearest approach to bony structure being an occasional infiltration of the fibrous or cartilaginous tissues with bone salts.

True *fibroma* of the testis seems to be of almost equally rare occurrence. Mr. T. Holmes has recorded one instance of a large fibrous growth of the tunica vaginalis, the size of a cocoanut, close above the testis, of thirty-three years' growth; and Mr. Haward exhibited at the Pathological Society of London, in 1872, a specimen of genuine firm fibroma springing from the tunica albuginea, and projecting into the testicle of a man, aged eighty-one. In this instance, the tumour—which closely simulated a chronic enlargement of the testis of eight years' duration—was removed with the testis by Mr. Pollock, at the patient's request, and on examination it was found to possess all the ordinary characters of dense fibroma of other organs.

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* "Trans. Path. Soc.," vol. vii. p. 241.

Loose bodies are occasionally found in the tunica vaginalis, similar to those occurring in joints. They are, in all probability, masses of exuded fibrine, and have, at least in the majority of cases, no claim to the epithet cartilaginous, which is often given to them on account of their external appearance. Calcareous matter is sometimes deposited in them; and Mr. Curling has observed the presence of laminæ similar to those of bone.

Dermoid cysts, sometimes containing besides well-formed skin with sebaceous follicles, hair and bone and teeth, are met with occasionally in the testicle, just as they are in the ovary, and they have been explained generally on Geoffroy Saint-Hilaire's theory of "fœtal inclusion," the germs of a second embryo being supposed to be included in the testicle of the first. But as before stated (p. 157), and as has been remarked by Professor Humphry on this special point, "It may well be doubted whether such a process ever takes place." * They are far more likely to be the result of departures from the natural formative processes in the testicle at a period when the developmental forces are in activity, and when morbid products are more prone to be composed of natural structures than they are when occurring at subsequent periods of life. Some of these dermoid cysts have a very complex constitution, containing several kinds of tissue, not always epidermic in character. Such growths must be called *teratoid* tumours. This is one of the rare situations where striated muscle has been found as a new growth. Such tumours also occur in the scrotum.

Certain fibroid masses, reported by M. Jarjavay † and others as growing from the fibrous envelope, and partly invading the testicle, and partly sprouting out as a superficial form of benign fungus, are probably of an inflammatory origin, and partake of the characters of so-called "granulation tumours."

Varicocele.—Morbid dilatation of the spermatic veins constitutes varicocele, a condition so common, and, as a rule, with few exceptions, associated with symptoms so trifling, as hardly to merit the name of a disease. Caused by many conditions,—as the long and winding course of the veins, their free anastomoses, and their liability to frequent compression in their course through the inguinal canal,—the enlarged veins in their turn may become a cause of some wasting of the testicle. When much dilated, and with walls thickened by the fibroid development present in most cases of varicose veins, the vessels form a swelling, feeling like a bunch of worms below the testicle, and reaching up towards, but rarely invading, the cord; to be distinguished from hernia by its peculiar feel, and by its gradual decrease in size on the patient's assuming a recumbent posture, to return on his standing up, the inguinal canal being free, and by the history of the slow course of the complaint. The left veins are oftener affected than the right, partly in consequence of accumulation of harder fæcal matter in the descending colon than in the ascending, partly on account of the left spermatic vein opening at right angles into the renal, and partly, perhaps, from the lower position of the left testis. When the varicocele is slight, it does not impair the nutrition of the testis; but, when large, it occasions very marked atrophy—doubtless, in consequence of the increased venosity of the retarded blood.

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* "Diseases of the Male Organs of Generation." By G. M. Humphry, M.D., F.R.S., in Holmes's "System of Surgery," 2nd edition, vol. v. p. 147.

† "Archives Générales de Méd.," 4ème série, t. xx.

Fatty tumours are occasionally developed in the spermatic cord, and are to be distinguished from encysted hydrocele of the cord by their doughy inelastic feel. Like the cysts, these tumours are often movable to a limited extent, and so simulate hernia. These fatty tumours may be complicated by the presence of sarcoma tissue together with the fat. This was the case in a specimen of recurrent fatty tumour of the cord, shown by Mr. Curling at a meeting of the Pathological Society of London in 1867. Microscopic examination of the tumours removed discovered the presence of spindle-cell growth, resembling "fœtal connective tissue," in the peduncle, which had been cut through at each operation, and from which, doubtless, the recurrent tumour had been developed.

MORBID CONDITIONS OF THE SCROTUM.

It may be well to notice here the morbid conditions of the scrotum, as a portion of integument which has somewhat peculiar relations. It is one of the most frequent situations of *elephantiasis* —a disease which M. Cazenave regards as essentially connected with inflammation of the lymphatics of the part. This produces the most enormous enlargement of the part—such that the mass has been known to weigh two hundred pounds more than the weight of the rest of the body. The epidermis, the corium, and the subcutaneous areolar tissue, are all, especially the latter, greatly hypertrophied. The areolar tissue is converted into a large mass of fibrous material, infiltrated with an albuminous and fibrinous fluid. Its areolæ are much enlarged in some parts; the testes remain sound; the spermatic cords are elongated several inches, owing to the testicles being dragged down, but are not otherwise diseased. Hydrocele sometimes occurs. When the disease is confined to the scrotum, and the enlargement becomes very great, the penis is drawn in, and ultimately disappears, while the elongated prepuce is continuous at a navel-like opening in the skin of the surface of the tumour.

Common hypertrophy of the integument of the scrotum sometimes occurs; in this there is no alteration of the subcutaneous tissue.

Epithelioma is the common form in which cancer appears in the scrotum, constituting what is generally called *chimney sweeper's cancer*, on account of its being apparently produced by the contact of soot. It is very remarkable that the disease may not appear till many years after the person has ceased entirely to be in any way exposed to the influence of soot. Mr. Curling mentions a case in which a man, after having been a sweep, went to sea, and led a sailor's life for nineteen years before the disease made its appearance. It is, of course, possible that, in these and similar cases, the cancerous development and the sooty employment were mere coincidences. The disease advances by invading the adjacent tissues, and thus produces fearful ulceration, extending even to the groin and thigh, and destroying life by perforating the coats of some of the large vessels. Wide diffusion of the disease throughout the body is happily rare, but the lymphatic glands in the groin not unfrequently become affected. Histologically, scrotal cancer consists of coarse squamous epithelium, in masses imbedded in a slight stroma of connective tissue, and having large "bird's-nest" collections of epithelial scales in rich abundance.

A case of *melanotic cancer* of the scrotum has been observed by Mr. Curling, and other forms of new growth, including fibroma and sarcoma, have been occasionally met with in this region. Dermoid cysts also occur here.

ABNORMAL CONDITIONS OF THE VESICULÆ SEMINALES.

Chronic catarrhal inflammation not uncommonly attacks the vesiculæ, causing tumefaction of their mucous membrane, secretion of unhealthy mucus, dilatation of the cavity, and thickening of its parietes. Ulcerative destruction of the mucous membrane occasionally takes place, the result of which may be thickening of the parietes (the morbid action having subsided), or perforation, with formation of abscess in the adjacent parts.

The vesiculæ seminales are occasionally affected by scrofulous inflammation, in which the tubes are distended with the fatty products of the epithelial proliferation, whilst the intertubular connective tissue is but little altered.

Professor Humphry has also described an enlargement, with induration of these bodies in old age, producing symptoms simulating those of enlarged prostate, and presenting the appearances of scirrhous carcinoma. In such cases, the seat of the change is the connective tissue, which is extensively indurated and thickened without much alteration being noticeable in the tubes. Scarce anything is known respecting *cancer* of these parts, except that they only suffer secondarily from extension of adjacent disease.

Hydrocele of the seminal vesicle has been described, and Professor N. R. Smith, of Baltimore, has recorded * a remarkable case, in which the collection of brown serous fluid amounted to twenty pints, and gave rise to all the symptoms of retention of urine, the bladder expelling its scanty contents every half hour, whilst a large fluctuating tumour distended the belly, and bulged into the partly obstructed rectum.

ABNORMAL CONDITIONS OF THE PROSTATE GLAND.

The prostate is imperfectly developed when the organs of generation are so. Its size is diminished in some cases, when the testes are atrophied; it is then rather consolidated in texture.

Hypertrophy of the prostate is an affection of advanced life, about one man in three having some enlargement of the part after sixty years of age, whilst in about twelve per cent. of men living beyond this age the enlargement becomes notable, so as to be the source of more or less trouble to the patient. In exceptional cases the prostate may attain a size so great as to displace the other pelvic contents, as in a case recorded by Sir Henry Thompson, in which a prostate weighing twenty ounces pushed the bladder nearly up to the umbilicus, even when empty. The condition comes on quite imperceptibly, as one of those changes whose existence is unknown till their secondary effects begin to be produced. The enlargement may take place in all the dimensions of the gland, so that it expands laterally, downwards, and upwards. Most often one lateral lobe is enlarged more than the other, which occasions a deviation of the urethra to the other side. It is said that the left is the one most commonly affected, and that it also frequently projects more than the right towards the cavity of the bladder. This, however, is by no means an invariable rule. The so-called third lobe is commonly enlarged more or less when the lateral lobes are so, but may attain a great size without any corresponding hypertrophy of the lateral. It forms a kind of pyramidal elevation, projecting into the cavity of the bladder, and causing the urethral orifice to be raised, and in some measure blocked up. The enlarged middle lobe has been found of the size of a small orange, more often it does not exceed that of a walnut; its surface may be smooth or nodulated. The hypertrophy of this part, when considerable, has the effect of throwing the neck of the bladder forward, and increasing the depth of its lower region, so that calculi may lodge behind and below the prostate in its cavity. The canal of the urethra becomes lengthened in its prostatic portion; sometimes divided into two channels by the projection of the middle lobe; or is tortuous from being curved to one side; or, in consequence of its vesical orifice being raised, describes a curve whose convexity is downward. The pressure on the rectum causes flattening of that channel, and more or less uneasiness in it, and may, perhaps, occasion hæmorrhoids, or prolapsus ani; in the same way it irritates the vesiculæ seminales, and induces thickening of their walls; pains felt in the parts to which the sacral nerves are distributed may be dependent on direct contact of these nerves with the enlarged gland, or on a reflected stimulus conveyed to their place of origin along the compressed vesical and hæmorrhoidal filaments.

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* "Lancet," Oct. 19th, 1872.

The most important result, however, of the enlargement, is the obstacle which it causes to the complete evacuation of the bladder. This depends partly on the narrowing of the urethra, and partly on the circumstance that a certain quantity of urine is always contained in the lower part of the bladder, below the elevated orifice of the urethra. In some cases the urethra, instead of being narrowed by compression, so as to appear like a slit on a transverse section, is considerably dilated, so that the prostatic sinus may contain two or three ounces of urine. An enlarged prostate is often indurated, so that by older writers it has been called scirrhus. The retained urine decomposing, causes irritation and inflammation of the bladder, with all its results, such as we have already described. Moreover, as Mr. Coulson describes it, the enlarged middle lobe becoming broader, and raising up a transverse fold of mucous membrane, which passes off on each side to the lateral lobes, constitutes a kind of valve, which is pushed before the urine in every attempt that is made to void it, "and closes up the opening till the cavity of the bladder is very much distended; then the anterior part of the bladder being pushed forward, and the tumour being drawn back in consequence of the membrane of the posterior part of the bladder being put on the stretch, the valve is open, so that a certain quantity of water is allowed to escape, but the bladder is not completely emptied."

The enlargement in these cases of hypertrophied prostate is due mainly to an increase in the connective and plain muscular tissue forming the bulk of the gland. With this the crypts of the glandular structure may be also dilated. In certain cases the development of the fibro-muscular tissues takes the form of separate pea-like tumours imbedded in the substance of the gland, and analogous in all respects to the fibro-muscular masses forming the "fibroid tumours" of the uterus.

Acute inflammation may attack the prostate, commonly as the result of extension of gonorrhœal inflammation; it may go on to suppuration, or cause chronic enlargement, or an irritable condition of the gland, with increase of its secretion. When abscess occurs, there may be one or several; a single one large enough to contain half a pint of matter, or small and numerous foci, so that the gland appears riddled with holes. The abscess may open into the bladder, into the prostatic sinus of the urethra, into the rectum, or, externally, into the perinæum.

Ulceration of the prostate scarcely occurs, except in some cases of chronic hypertrophy. It occasions severe suffering, and the admixture of blood with the urine.

Tubercle is not of frequent occurrence in the prostate; it is generally co-existent with tuberculosis of other parts of the generative apparatus, and of the lungs. It may form a single large mass, or numerous small separate ones. As the tubercles soften and disintegrate they give rise to abscesses, which pursue the same course as those of inflammatory origin.

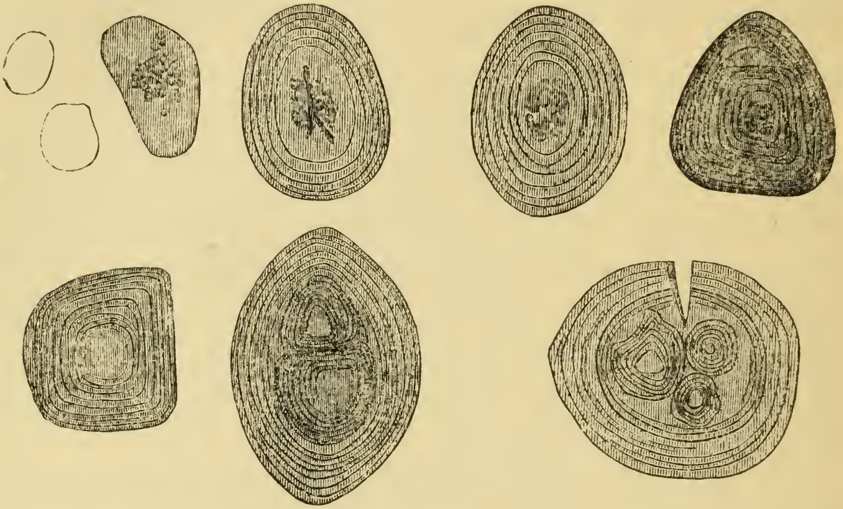
Cancer is rare in the prostate; five cases of it only were met with by M. Tanchou among 8,289. The scirrhous species has been scarce ever observed. Encephaloid, either primary or extending from the bladder, is almost the only form that occurs. It causes considerable enlargement of the gland, and may also perforate the mucous membrane of the bladder, and vegetate in its cavity, filling it up so completely as to give rise to the idea of the viscus being distended with urine. The disease occurred in a patient of Mr. Stafford's, at the age of five years, and in one of Mr. Solly's at three. After childhood, however, the disease is not encountered until after middle life, no authentic case being recorded between the ages of eight and forty-one.

Cysts are in extremely rare cases formed in the prostate, as Mr. Adams thinks, by closure of the outlets and dilatation of the cavities of follicles.

Concretions, in greater or less number, are of almost constant occurrence in the prostatic cavities; they may often be seen, on making a section of the gland, as reddish yellow grains.

The larger, fully formed ones, show under the microscope a beautiful laminated structure, and resemble a good deal a section of a lithic acid calculus. Their form varies very much; in the smaller it approaches the oval or circular—in the larger, it is more polygonal, or triangular. They are not unfrequently pale or colourless. They originate in large oval vesicles, formed of a well-marked homogeneous envelope. These appear to increase in size, while concentric laminæ are formed in their interior, whose interspaces are occupied by a finely mottled deep yellow or red matter. A central cavity is almost always left within the last-formed laminæ. Deposition of organic matter may take place in some cases exterior to the original envelope, but in most it appears to be within. Concretions of older date seem to lose the beautiful definiteness of their structure, and tend to disintegrate. The contents of these semi-organized formations appear to be earthy matter (phosphate, with a little carbonate of lime), tinged by the ordinary yellow pigment which is so often derived from the blood. We do not think they are developed from the ordinary epithelial particles of the gland, but that the original vesicles are cells of a particular kind, which are produced from organic exudation upon the mucous surface, and fill themselves, as their growth proceeds, with successive deposits of materials, which are probably poured out when the gland is the seat of vascular excitement.* It is most probable that in ordinarily healthy states these concretions undergo solution at an early period of their existence, yielding up their contents to form part of the secretion of the gland. But if this does not occur, and they go on increasing in size, they become the nuclei of, or are developed into, prostatic *calculi*. They are sometimes very numerous, as many as fifty or sixty having been found in an atrophied dilated prostate, which has, in consequence, when examined *per rectum*, given the sensation of a bag of marbles. The calculi sometimes cohere together, and form a large mass, projecting into the membranous portion of the urethra, which becomes in consequence much dilated. A remarkable case of this kind has been recorded by Dr. Herbert Barker,† in which twenty-nine calculi, weighing together 1,681 grains, were cemented together so as to form a single concretion, which was nearly five inches long, and of an elongated pyriform shape. The surfaces of these calculi are faceted from mutual pressure; they are of a whitish or reddish colour, of porcellaneous lustre and hardness, with a radiated,

FIG. 178.



Prostatic concretions magnified. (Dr. H. Jones.)

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* The organic base of these concretions has the same shape and arrangement as the mineral constituents, and is apparently albuminous. Their formation seems to be an admirable instance of the process called "molecular coalescence" by Mr. Rainey, which we have referred to in the case of urinary calculi. Mr. Rainey has shown that the carbonate of lime, when formed by double decomposition in an albuminous, mucous, or gummy medium, assumes the form of concentric bodies much resembling the prostatic calculi and also certain urinary calculi of the horse. Mr. Vandyke Carter has seen similar spheroidal forms in phosphates.—ED.

† "Transact. of Prov. Med. and Surg. Association," vol. iii. 1846.

laminated, or compact structure. Lassaigne's analysis gives the following as their composition: basic-phosphate of lime 84·5, carbonate of lime 0·5, animal matter 15. The smaller calculi often escape into the bladder through the dilated prostatic ducts; if they remain there they excite irritation of the mucous membrane, and deposition of phosphates upon their own surface. A coating of lithic acid has sometimes been formed on a large calculus remaining in the prostatic portion of the urethra.

ABNORMAL CONDITIONS OF THE PENIS.

The penis is very imperfectly developed in some cases of normal condition of the other sexual organs, as well as when they are themselves imperfect. We have seen it extremely short and fissured in its whole upper surface in a case of *eversio vesicæ.* When it is very small, fissured below, and destitute of prepuce, and when at the same time the testes remain in the abdomen, and the scrotum is cleft, there results a considerable resemblance to the female conformation; or, if the penis is less atrophied, a pseudo-hermaphroditism. Atrophy of the penis, accompanied by obliteration of the cavernous textures, occurs, according to Rokitansky, together with atrophy of the testicles.

The penis is subject, like the rest of the body, to *inflammation*, which, affecting various parts, gives rise to special symptoms, and receives special names. Thus, "*chordee*," a peculiar and painful twist of the erected organ, may result from inflammatory thickening in the corpora cavernosa or spongiosum, or from the formation of a gummy tumour in the corpora cavernosa of a syphilitic. *Balanitis*, inflammation of the mucous membrane of the glans; and *posthitis*, inflammation of the inner surface of the prepuce, with resulting ulceration and even gangrene in neglected cases, are amongst the accompaniments of venereal disease.

The *vesicles of herpes* sometimes form on the prepuce, on its mucous or cutaneous laminæ.

Venereal Sores.—More grave by far than these lesions, however, are the special ulcerations met with as results of impure sexual intercourse. These ulcers, or *chancres*, as they are commonly termed, are found on the glans, in the sulcus behind the glans, on either surface of the prepuce, and even within the urethra—although probably seldom more than a few lines from the urethral orifice. They are of two kinds—(1) The *hard*, or Hunterian chancre, the initial manifestation of the constitutional disease of syphilis; and (2) The more common *soft*, non-infecting venereal sore; and to either of these, but especially the latter, may be added, in certain depressed conditions of the system, an irregular sloughing action converting the indolent sore into a rapidly-extending, foul ulcer, then styled *phagedenic*.

The initial manifestation of syphilis, according to Mr. Berkeley

Hill, may take three forms. 1. *The desquamating papule*, a reddish-purple solid elevation, covered by a few thin epithelial scales, and gradually spreading from the size of a pin's head to that of a sixpence or a shilling. 2. *The superficial erosion*, a moist, slightly raised surface much resembling the "mucous patches" of the later stage of the disease, and met with in similar positions, as under the foreskin in the male, and the inner nymphæ in the female. 3. *The indurated ulcer*, beginning as a hard papule, but gradually assuming its typical appearance of a sore with a hard base, feeling to the touch like a cup of gristle inserted in the skin, with a surface covered by a scanty adhesive yellow discharge, and bounded by sloping rounded edges, the induration extending somewhat beyond the immediate margin of the ulcer. This initial manifestation of syphilis may remain for from three or four weeks to as many months, when, under treatment, the induration and other signs gradually disappear.

The *soft chancre*, or non-infecting venereal sore, is distinguished from the syphilitic lesion by the absence of marked induration, by its sharply-cut margin giving it the appearance of being punched out of the skin, and by its spongy, worm-eaten base, bathed with a foul auto-inoculable pus. As a result of this last important property, it usually happens that these sores are multiple, especially in women on the opposite surfaces of the labia pudendi, and around the free margin of the prepuce in men.

The anatomical characters of the induration tissue underlying a syphilitic ulcer, although closely resembling those of the gummy tumours subsequently forming in the skin, the viscera, and elsewhere, and hence deemed by Wagner a structure peculiar to syphilis—syphiloma—probably differ in no material respect from the appearance presented by any form of chronic inflammatory lesion. Beneath the thickened cuticle and enlarged papillæ, a dense layer of fibro-corpuscular growth is seen, the corpuscles resembling leucocytes, and separated only by a slight granular substance, or enclosed in the meshes of a definite fibroid stroma, which in some instances is of considerable density. A structure precisely resembling this is met with wherever slow irritative processes have been long at work, as in an old indurated pile, or about the cavities of lungs destroyed by chronic phthisis.

The term *ulcus elevatum* has been applied to a species of sore unconnected with syphilis; probably a variety of what is now called the soft sore.

Phymosis is the name given to such a contracted and elongated prepuce as cannot be drawn back over the glans. This is usually a congenital defect, and may be present to an extreme degree. We have seen a man whose preputial orifice was from birth no larger than a small pinhole, and in whom circumcision was required in middle life to enable the penis to perform its functions. Sometimes such a condition results from psoriasis of the prepuce, or from the contraction following the healing of venereal ulcers.

Paraphymosis is the opposite condition, in which a tight prepuce, having been drawn back, constricts the neck of the glans, from having itself become thickened, and thus occasions a distended state of the glans, and even mortification unless the stricture be removed. Phymosis is apt to give rise to attacks of balanitis, from the accumulation of the secretions of the coronal follicles.

New Growths.—Warty vegetations sometimes form on the glans, or on the inside of the prepuce; they are commonly the result of the irritation of foul urethral discharges, and are capable of being cured effectually by removal.

Cancer may affect any part of the penis, but is most frequent on the glans and prepuce. Cases of *sarcoma* (in two instances melanotic, and made up of spindle cells) have been recorded in this position. And still more rarely a *scirrhous* infiltration of the body of the penis has been observed, but by far the most common form of cancer here is *epithelioma.* The disease may originate as a warty excrescence, or as a pimple which discharges an excoriating fluid, scabs, and breaks out afresh, while induration advances at its base; or it may infiltrate the glans, so as to convert that part into an indurated mass. After a lengthened interval ulceration supervenes and sometimes spreads with rapidity, destroying all neighbouring tissues in its march, and causing at times great pain. In rare instances the warty vegetations—which can with difficulty be distinguished from simple growths at first—spread down the urethra, but more commonly the superficial structures suffer most. The histological characters are those of ordinary squamous-cell epithelioma. Secondary growths, except in the adjacent glands, are not of common occurrence. Phymosis and the irritation attending upon it, seem to act as exciting causes; advanced age as predisposing, whilst, in some few instances, direct contagion from a cancerous uterus has been alleged to be present.

THE PATHOLOGICAL ANATOMY OF THE FEMALE ORGANS OF GENERATION.

CHAPTER XL.

THE EXTERNAL ORGANS OF GENERATION.

In the labia, suggillations are frequently met with as a result of external violence, or after parturition; the effusion from a violent injury may give rise to very considerable tumefaction, which must not be confounded with varicose swellings. When the consequence of childbirth, it generally affects the left labium,* and occurs more frequently in primiparæ than multiparæ. The swelling in either case has been known to attain the size of a fist, or a child's head. It presents a tense, smooth surface, with a livid colour. Varicose veins of the labia may also acquire a very considerable size; but the slow increase of the tumour, and the vermicular character of its contents, together with the ready compressibility of the swelling, will determine the diagnosis. Varicose swellings, too, occur during the course of pregnancy, and, though sometimes very considerable, do not generally cause any impediment to parturition, as they are external to the vulva. Cases, however, are recorded of their sudden laceration during parturition, and of a consequent fatal issue. The hæmorrhagic tumour disappears spontaneously, or in consequence of treatment, but exceptionally the swelling persists, probably becoming encysted, and may then be borne for an indefinite period. A case is related by Mauriceau,† in which a tumour, originating in this way, existed for twenty-five years. A ready explanation of the common occurrence both of large blood extravasations and varicose swellings is to be found in the fact that the vaginal veins are destitute of valves.

A form of atresia vaginæ, not unfrequently met with amongst infants, and occasionally suffered to persist until adult life, is caused by adhesion of the labia pudendi. The closure in these cases is usually not complete, being limited to the posterior half of the orifice, and, when detected in infancy, may generally be remedied. If, however, owing to the neglect of the surgeon or the parents, the adhesion is suffered to remain, a firm membranous septum is formed which may need operative measures of an active kind.

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* "Kilian, die Geburtslehre," &c., vol. ii. p. 517, 1840. Frankfurt.

† "Observations sur la Grossesse et l'Accouchement des Femmes," Paris, 1695, Obs. 29.

Inflammation.—Inflammatory affections of the labia may arise from internal and external causes, and exhibit the various forms of inflammation met with in other superficial textures. Eczematous and aphthous inflammation, as a result of derangement of the digestive organs, of pregnancy, of a want of cleanliness, or of sexual over-indulgence, are common. Eczema is characterized by the appearance of a vesicular eruption scattered over the inner or outer surface of the labia. The vesicles break and scab, and they are the source of much of the pruritus to which females are subject.

The loose cellular tissue, occupying the interval between the external and internal labia, especially favours œdematous swelling, and, when the inflammation bears a phlegmonous character, extensive sloughs form. Instances of this in early life are recorded by Mr. Kinderwood,* who witnessed an epidemic at Manchester, marked by great fatality.

The mucous crypts, especially the aggregation lying on each side of the vestibulum, and termed by Bartholinus the female prostate, are liable to inflammation from catarrhal, herpetic, syphilitic, or other causes, resulting in chronic ulceration or tedious discharges. Even young children are frequently liable to simple or benignant inflammatory affections of these parts, giving rise to much irritation and muco-purulent secretion—a circumstance with which it is necessary to be acquainted, as popular prejudice is only too prone to attribute it to contagion. Abscess in Bartholini's gland is distinguished from abscess in the cellular tissue of the labium majus, or from ischiorectal abscess, by its being limited to the furrow between the labia and by its pointing on the inner side of the nympha, or in the furrow.

Warts, due generally to the results of impure intercourse, but not necessarily associated with a syphilitic taint, frequently disfigure the vulva. They resemble in all respects the similar warty excrescences commonly met with about the prepuce of men suffering from urethral discharge. They may be pedunculated, and no larger than small peas, or many warts conglomerating, both labia may be converted into a tumour of great size, whose surface is shaggy, with firm pale coarse papillary projections bathed in an offensive and highly irritating discharge. When such a mass is removed, the subcutaneous connective tissue is found to be little altered, the changes being limited to an hypertrophy of the skin, the papillæ being specially the seat of the overgrowth.

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* "Med.-Chir. Trans.," vol. vii. p. 84.

Due in the first place to the irritation of foul vaginal discharge, the thin fluid exuding from these warts is apt to cause fresh warty growths to spring from the skin soiled by it. Hence the most frequent seat of these excrescences is the lower part of the vulva, the perinæum being also a common position, and the clitoris and adjacent parts being less often affected; but they may be met with in all parts of the external sexual apparatus.

Mucous tubercles, or mucous patches, or "*plaques muqueuses*," are growths allied to warts in their nature, but differing from them in that they are only met with in patients suffering from syphilis. They commence as flat, circular, or oval elevations, of a rosy or purple colour, having a diameter of a quarter or half an inch, bathed in thin, offensive, highly contagious secretion, and situate usually about the vulva or anus, or mouth; but they may occur wherever two perspiring surfaces are in contact, or upon mucous surfaces, as the tonsils. The small patches are apt to coalesce, and form large irregular elevations, especially on the perinæum, and they are usually so painful as to attract notice early. They are frequently present in syphilitic women, and are often almost the only signs of the disease present.

Tumours.—Encysted tumours of slow growth affect the labia, and are probably due to an obstruction in the first instance, and subsequent distension of one or more of the follicular structures. They consist of a membranous envelope, containing a transparent, glairy fluid; and only prove a source of inconvenience after they have attained a large size.

Other tumours are described as occurring in the pudenda, independently of the hypertrophy resulting from chronic inflammation. Sir Charles Clarke has described a variety under the designation of the oozing tumour of the labium, which is chiefly characterized by a profuse discharge from the muciparous follicles. It is but slightly elevated above the skin, and has an irregularly nodulated surface. It occurs in persons advanced in life, endowed with general obesity, and in whom the labia are enlarged. Erectile and fibroid tumours are also met with in this part of the system. A remarkable specimen of the latter, successfully removed by operation, is preserved in the Royal College of Surgeons of England (No. 2,715). It weighed upwards of eleven pounds, and is six inches in diameter. It is covered with healthy skin, and consists of a pale and compact, but soft and elastic, tissue, traversed in some parts by irregular, shining fibres, and in others having several small oval cavities in it. The patient was thirty years of age, and the tumour had been growing for many years.

Hypertrophy.—The vulva is also subject to enormous enlargement from elephantiasis of the skin, an affection rarely witnessed in this country, but epidemic at Barbadoes. In this disease the labia may be so greatly hypertrophied as to reach to the knees. A less rare form of enlargement of labia and nymphæ is due to an increase of the subcutaneous fat and connective tissues; but this variety seldom attains any very great size.

In new-born infants the nymphæ normally project beyond the labia majora, and, in some wild tribes, the custom exists of inducing their elongation by artificial means; this is said to be the case among the Bushmen and the Kamschatdales. Among the Arabs and Copts circumcision of females prevails, which consists in removing a portion of the elongated nymphæ.

The enlargement of the nymphæ has been set down to an abuse of sexual indulgence, but this is, probably, as incorrect as the same statement has been shown to be with regard to hypertrophy of the clitoris. This rudimentary penis excites no attention, unless enlarged beyond its normal proportions. It is capable of assuming the most extravagant size. Some of the cases of hermaphrodism that are on record may be explained by a reference to congenital hypertrophy of the clitoris. The largest specimen that we have met with is preserved in the Museum of the University of Bonn. It is fourteen inches in circumference, and weighs eight pounds. Mr. Safford Lee quotes several instances of similar hypertrophic enlargement. Parent Duchâtelet met with enlarged clitoris in only three cases of 6,000 registered prostitutes in Paris. Dr. Ashwell, in his remarks on the subject, expresses his concurrence with the last observer as to there being no necessary connection between an habitual sexual indulgence and the permanent increase of the clitoris. He adds, that he has often been struck with the integrity of the external genitals in prostitutes, while the uterus and ovaries have been bound in all directions by bands of false membrane. Cancer of the clitoris is rare, but we have seen two instances of epithelioma in this position; and in one case, which terminated fatally, the disease involved largely the inguinal glands on both sides, and appeared also in characteristic nodules in the heart—all these secondary growths having the typical characters of squamous-cell epithelioma.

We allude to the urethra at present, only, to speak of certain affections of the orifice, which opens beneath the clitoris, into the vestibular portion of the vagina. The very large crypts and sebaceous follicles surrounding this sensitive point are the frequent seat of blennorrhoic and other forms of inflammatory action.

The so-called *vascular tumours*, or urethral hæmorrhoids are not uncommon in early middle life. These small, bright, red excrescences usually spring from the urethral orifice, or from the floor of the urethra close to the orifice, and have been already spoken of.

THE HYMEN.

The valvular fold of membrane which protects the virginal vagina, the hymen, which is commonly ruptured when coition is first completely effected, has been a subject of much discussion by medical jurists, as its absence has been regarded as an unequivocal sign of defloration, or its presence as a proof of the unimpaired virginity of the individual. Neither position is absolutely correct; for the best authorities are agreed that, on the one hand, it may be destroyed by ulcerative absorption, or, on the other, that it may persist, not only after coition, but even after parturition. The latter fact is corroborated by the testimony of Merriman, Nägelé, Ramsbotham, and others. Other deviations from the normal state of the hymen are the cribriform perforations that it exhibits; or it surrounds the entire introitus vaginæ, leaving a central circular orifice, or it entirely occludes the passage. The latter circumstance is not likely to be discovered, as other atresiæ of the external orifices are, early in life. With the approach of puberty it will induce much inconvenience from the mechanical retention of the menstrual discharge, and, unless discovered and rectified, will be the source of serious disturbance. The hymen is sometimes found much indurated, and of a cartilaginous consistency, and even osseous deposits have been met with in it. The hymen, after it has been ruptured, is partially, if not entirely, absorbed. The carunculæ myrtiformes, which have been generally looked upon as the remains of the hymen, are now regarded by many authorities as normal formations that are not associated with lesion of the hymen.

ABNORMAL CONDITIONS OF THE VAGINA.

The vagina presents very considerable varieties of conformation and size within the normal limits of health, differences depending upon the age of the individual and the effects of cohabitation or childbirth, or the absence of these influences. A congenital closure of the passage may, independently of an imperforate hymen, or adhesion of the labia, convert the vagina into a cul-de-sac, a lesion which can scarcely be attributed to anything but intra-uterine inflammation, if the uterus be present. A remarkable instance, which appears to have been an arrest of development, is detailed by Dr. Boyd,* where, in a female, aged seventy-two, who had been married, though necessarily without issue, the vagina terminated in a cul-de-sac, about half an inch deep, beneath the orifice of the urethra. There was no vestige of a uterus, nor any Fallopian tubes; the right ovary was natural, and attached by a loose ligament to the bladder; the left ovary was abnormal, but similarly connected with the bladder. A multiplication of parts is, perhaps, more frequently met with, and is produced by the formation of a septum, which is more or less complete; it may extend through the entire length of the vagina, or only partially divide it. A remarkable specimen of this malformation was exhibited by Mr. Birkett before the Pathological Society;† the vagina of a married

* "Med.-Chir. Trans.," vol. xxv. p. 187.

† "Report, &c., 1847-48," p. 295.

woman, who had never borne children, and had died of pneumonia and pericarditis, was completely divided in the mesial line by a strong, dense, fibrous septum, extending from the external opening to the uterus. Thus two vaginæ existed; each vagina led to a distinct os uteri, both of which were small; the neck of the uterus was rather longer than usual, the body smaller; the uterus itself was nearly divided into two cavities, by a septum in the mesial line.

Occlusion, or stricture of the vagina, sometimes occurs as a result of external injury or of cicatrization of ulcers. The rigidity or laxness of the walls varies much in different subjects, according to the general habit and the amount of secretion from the glandular apparatus surrounding the vagina. The great capability of the vagina for extension is best shown in parturition; hence it is not to be wondered at that prolonged uterine or vesical disease should induce a very lax state of the mucous membrane of the vagina, which, as it often does, becomes a source of extreme distress and inconvenience to the patient. In old women we often meet with this relaxed condition, which may amount to a complete prolapsus. The anterior wall is particularly prone to be thus affected. Dr. Golding Bird * has pointed out that this lesion gives rise to a fœtid, phosphatic, and mucous state of the urine in elderly females, owing to an accumulation of the urine in the prolapsed bladder lying in a pouch of the anterior vaginal wall. He shows that it may be the source of great irritability of the bladder and incontinence of the urine, which is best relieved by frequent catheterism, so as entirely to empty the bladder. In prolapsus of the uterus the mucous membrane of the vagina is necessarily dragged down with the descent of that organ.

Injuries.—The vagina and the external organs are exposed to mechanical injuries of various kinds, and, in certain medico-legal questions, it requires care to determine their exact nature, as well as to avoid confounding the menstrual discharge with hæmorrhage resulting from injury. Parturition frequently gives rise to laceration and severe contusions of these parts. The inferior portion of the canal, either from unusual rigidity, or from want of proper care on the part of the attendant, is apt to give way when the labour pains are at their climax; and the lesion may vary from a mere laceration of the fourchette to a rupture of the entire perinæum, from the vagina to the anus. It may happen as a very rare accident that the perinæum is perforated without the rent extending into the natural opening of the vulva. Laceration of the upper portions of the vagina also occurs to a varying extent, in conjunction with, or independently of, rupture of the uterus. A small laceration is not necessarily fatal. Ross † reports the case of a woman who was twice the subject of an accident of the kind, and each time recovered. This result is out of the question, when, as occasionally happens, the child escapes into the peritoneal cavity.

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* "Medical Times and Gazette," January 1st, 1853.

† Dr. Francis H. Ramsbotham gives this and other illustrative instances in his "Principles and Practice of Obstetric Medicine and Surgery," p. 603. London, 1841.

Vaginitis.—The mucous membrane of the vagina is very frequently the seat of inflammation; the commonest form is the catarrhal. In the first stage the passage is reddened, heated, and dry; this is followed by the secretion of a white, creamy mucus; or, if there be anything of a specific character, the discharge is more purulent or flaky.

The mucous secretion, which serves to lubricate the vagina in health, may be poured out in large quantities under certain conditions of plethora or weakness, and this already complex acid fluid, mingled with a free alkaline secretion from the glands lining the cervix uteri, forms a curdled white albuminous fluid, which flowing from the vulva constitutes *leucorrhœa.* This white discharge contains always more or less of altered epithelium, and it may be so abundant as to gravely affect the health of the sufferer. The cervix uteri is doubtless often implicated, but as the acid of the vagina is sufficient to neutralize the alkaline secretion of the uterus, the fact of the latter being frequent and copious is masked; hence the discrepancy of the opinions of various authors on the subject of the source of leucorrhœa. The external surface of the os uteri, according to Dr. Tyler Smith, yields a secretion of the same character as the vagina itself. In both, eczematous vesicles are frequently met with, which the same author regards as identical with the ovula Nabothi, which, by some, have been interpreted as obstructed follicles, but Dr. Tyler Smith asserts that they are often found in situations where mucous follicles cannot be detected.

False Membranes.—Rokitansky and Förster describe exudative, or croupous and diphtheritic processes, occurring in the vaginal mucous membrane primarily, but more frequently in conjunction with a similar disease of the uterus, in the shape of puerperal disease. Similar pseudo-membranaceous disease is said also to occur in the course of typhus, pyæmia, cholera, and the exanthemata; but these affections are probably rare in this country. Dr. Barnes states that diphtheria of the vagina occurs especially in lying-in hospitals. He also mentions a form of vaginal inflammation, in which the surface is covered with pellicles or flakes consisting almost entirely of agglomerated epithelial scales, and distinguished from the diphtheritic false membrane by being white and brittle. For this affection, which is not usually attended by febrile symptoms, he suggests the name *pseudo-diphtheritis.* A complete epithelial false membrane, like the dysmenorrhœal false membranes from the uterus, is preserved in the Museum of St. Thomas's Hospital.

Gonorrhœal vaginitis is difficult to distinguish from the simpler form; but, according to Dr. Barnes, the redness is much more intense, there is copious muco-purulent secretion of yellowish or greenish tint, and it affects more especially the fundus of the vagina, with some implication of the vaginal portion of the uterus.

A chronic thickening of the vaginal mucous membrane is occasionally met with. The catarrhal and the syphilitic ulcer also affect this part. Gangrene sometimes results from the effect of parturition, or the contusion caused by rough manipulation. The cicatrix that results from the healing of a slough is occasionally an impediment at subsequent deliveries; a puckering of the vaginal membrane, and consequent diminution of the passage, having taken place.

Tumours and New Growths.—The vagina is not often the seat of morbid growths. Polypi and encysted tumours are the varieties that most frequently affect this situation. The posterior part of the vagina is stated to be the ordinary seat of polypoid growths. An instance given by Mr. Curling, in the "Reports of the Pathological Society,"* forms an exception to the rule. Here the solid tumour, which was removed from a woman, aged forty-five, grew from the upper part of the vagina, to which it was attached by a broad peduncle, which commenced just behind the meatus of the urethra, and extended backwards towards the uterus about two inches and a half. The structure of the polypi varies in character; they may be fibrous or mucous—the fibrous being the least frequent. They vary equally in size, from a trifling projection to growths several pounds in weight.

The encysted tumours of the vagina originate in an obstruction of the follicles with which the upper and lower portions of the region abounds; they contain a glairy, transparent greenish or dirty-brown, albuminous fluid; and, though the source of irritation and inconvenience, are not productive of any danger, their correct diagnosis affording a speedy means of relief; but they have been repeatedly mistaken for totally different affections, such as prolapsus of the womb or the bladder, or for hernia.

Specimens of cancer affecting the vagina are preserved in most museums of pathological anatomy; they show that this part is commonly secondarily involved by an extension of the disease from the cervix uteri; "however, it may exist," to employ the words of Rokitansky, "though the latter is in a very undeveloped state, and even without it, in the shape of primary carcinoma of the vagina." The form of cancer present is doubtful, the microscopic appearances having been seldom recorded. We have ourselves seen one case of apparently obvious epithelioma of the floor of the vagina near the orifice, but there is a general belief that this form of disease is never met with in this position. Primary cancer of the vagina is undoubtedly extremely rare. It may be propagated from the rectum when the wall is destroyed by ulceration.

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* Vol. i. p. 301, 1847-48.

CHAPTER XLI.

ABNORMAL CONDITIONS OF THE UTERUS.

Congenital Malformations.—We have already given an instance (page 746) of an entire absence of the uterus—a malformation which need not affect the health of the individual. Rokitansky states that the occurrence is extremely rare, and that most of the cases in which the uterus appears to be absent may be resolved into a partial arrest of development only, and that, by careful examination, we may find, behind the bladder, one or two rudimentary bodies, in the proper fold of the peritoneum, which represent the uterus. An actual multiplication of the organ is equally rare; but it is not an uncommon thing to find a more or less complete attempt at the formation of a double cavity, which is manifestly the result of an arrest of development. The bilocular and horned uterus are the malformations alluded to; in the former, a more or less perfect septum extends through the uterus in the median line; in the latter, the organ presents the character of the uterus exhibited by certain mammalia, as the sheep, and is divided into two lateral compartments by a fissure, extending vertically downwards from the fundus. This may be explained upon the assumption of an imperfect union of the two rudimentary bodies from which the normal uterus is developed. Only one of these may arrive at maturity, and we shall then have a uterus consisting only of a single horn, or of one half; there will then, necessarily, only be a single Fallopian tube. The uterus unicornis, as well as the uterus bilocularis and bicornis, are capable of becoming impregnated; Rokitansky* details the particulars of an example of pregnancy in a rudimentary uterine horn, which terminated fatally by rupture and sanguineous effusion into the peritoneal cavity in the third month. There may be many intermediate conditions between the bicornute uterus and the organ with one horn so much larger than the other as to place it in the category of single-horned uteri.* When the two halves coalesce, the division which constitutes the malformation may vary considerably in amount; only a slight depression may be visible at the fundus in one case, so that the organ scarcely deviates from its normal condition; in another, the fissure extends so far down as to justify the appellation of double uterus, ordinarily bestowed upon the anomaly. An excellent instance of this is preserved in the Museum of St. George's Hospital (No. 104 of Dr. Lee's preparations). This preparation also illustrates what takes place after impregnation; while the ovum is received into one horn, which becomes duly developed with the growth of the fœtus, the other only sympathizes with it so far as to form a deciduous membrane, and thus to prevent the occurrence of superfœtation, but otherwise undergoes but trifling alteration or increase. Though impregnation and parturition are not necessarily fatal, these malformations seriously endanger the life of the patient—owing, as Rokitansky observes, partly to the want of the necessary dimensions of the part that undertakes the functions of the entire organ, partly to the obstacle opposed to the uniform development of the impregnated uterine half by the unimpregnated half. These circumstances favour laceration of the uterine parietes. Rokitansky also shows that the divergence of the cornua from the axis of the body causes an impediment in the act of parturition, while the expulsive power of the uterus is much reduced, by the absence, in the case of the uteris bicornis, of a true fundus.

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* "Pathological Anatomy," vol. ii. p. 277, Syd. Soc. Ed.

A highly interesting case of malformation of the uterus has been put on record by Mr. Croft, in the "Trans. Path. Soc.," vol. xix. The subject of the deformity lived only four days, and was brought to Mr. Croft for imperforate anus, the perinæum showing no indication of any anal aperture. After death a largely distended uterus was found bulging up from the pelvis, surmounted by two egg-like cornua. The cavity was full of viscid fluid—a mixture of urine and mucus—both bladder and rectum communicating with it by minute apertures. Descending from the partition between the horns of the uterus was a septum, complete save for a small oval foramen in the lower portion.

A more rare condition than those imperfect tendencies to single or double-horned uterus is an absence of the uterine orifice, forming one of the causes of retention of menstrual fluid after puberty.

HYPERTROPHY AND ATROPHY.

Hypertrophy and atrophy of the uterus are, in part, normal at the periods of puberty and involution; much tact is necessary to distinguish some of the morbid from the healthy conditions of the organ. The weight and dimensions of the adult uterus fluctuate in health considerably. Kilian[*] gives the following as the result of his measurements:—The entire length varies from twenty-four to twenty-six lines; the greatest breadth is eighteen lines; the greatest thickness, nine lines: the cervix is from ten to twelve lines long; its breadth from six to eight lines; its thickness from five to six lines: the length of the uterine cavity is twelve lines, its breadth nine lines; the greatest thickness of the fundus, five lines, of the sides, four lines, and of the cervix, three lines. After one or more births all these measurements increase from one-fifth to one quarter. The weight of the uterus varies from eight to twelve drachms, and may, after several pregnancies, amount to two ounces. With the aid of this table we shall be better able to determine whether we have to deal with a morbidly enlarged or diminished uterus. Either affection may involve the entire organ, or be manifested in a part only.

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* For more detailed accounts, and many interesting examples of these and other malformations of the uterus, the reader is referred to the valuable work of Kussmaul, "Von dem Mangel, der Verkümmerung und Verdopplung der Gebärmutter." Würzburg, 1859.

In certain cases, the uterus continues through the whole or part of adult life in the undeveloped condition met with in childhood, the various parts being perfectly formed—and, indeed, occasionally expanding at middle age—but the size of the organ not increasing as usual at puberty. This condition, which receives the name of "infantile uterus," may or may not be accompanied with ovarian defects, and is usually, though not always, associated with absence of sexual desire and the power of conception.

The infantile condition of the uterus is characterized by its small size, so that the uterus seems scarcely wider than the vagina, and by the disproportionate size of the cervix and comparative smallness of the fundus. This condition is sometimes seen in chlorosis, accompanied by defective development of the circulatory apparatus, as first pointed out by Virchow,[†] whose observations we have been able completely to confirm.

The term *atrophy* should not be applied to the persistent infantile condition of the uterus just noticed, but should be limited to those comparatively rare cases in which the considerable dwindling in size and activity met with at the climacteric period in advanced middle age is brought about at an unusually early period. Even in these cases, however, ovarian activity usually ceases with the premature involution of the uterus. The cervix is sometimes the chief seat of the atrophic changes, but it is more common for the entire organ to assume a fibrous, anæmic, attenuated appearance.

Certain of these instances of general atrophy of the uterus are due to what Sir J. V. Simpson styled "superinvolution" of the uterus after parturition.

Partial atrophy, caused by the pressure of ovarian or other adjacent tumours, or "eccentric atrophy," due to a thinning of the stretched uterine walls by an accumulation of contained fluid, are also occasionally met with.

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[*] "Die Geburtslehre," &c., von Dr. H. F. Kilian, vol. i. p. 92. Frankfurt, 1839.

[†] Virchow, "Die Chlorose," u.s.w. Berlin, 1872.

Hypertrophy is more commonly met with as a morbid state than atrophy; partly as an exaggerated expression of the normal condition, at certain periods of life, partly as the result of irritation set up by other morbid processes. These may consist in tumours, occupying the substance, or filling the cavity of the uterus; giving rise, eventually, to actual expulsive efforts, resembling labour pains, or to blennorrhœic affections of the mucous surfaces. The cervix is liable to be hypertrophied by itself; the labia may form a single tumefied ring, or present two tumours, lying parallel to one another, and separated by a transverse fissure. The first form is more likely to occur in women who have not borne children, and the second in those who have. The anterior is more frequently enlarged than the posterior lip. In the hypertrophy resulting from defective involution after delivery, the whole organ is involved, but seldom to any considerable extent, though the changes affecting the cervical portion are, of course, more apt to attract attention.

The cavity of the uterus may be morbidly diminished in consequence of inflammatory affections of the surrounding textures, or by malposition or curvation, and may amount to complete obliteration. An instance of obliteration of the cavity of the uterus is preserved in the St. George's Hospital Museum (Dr. Lee's Preparations, No. 161), in which the cervix remained patulous.

We shall have occasion to see that the os and the cavity of the uterus are frequently plugged up by secretions, but this must not be confounded with actual adhesion of the parietes. Strictures are commonly met with at the external and internal orifices of the cervix; they appear to be mainly due to inflammation of the mucous and submucous tissues of the parts.

UTERUS DURING MENSTRUATION.

The uterus swells considerably from the flow of blood which takes place at this time to the genital organs; its muscular substance becomes more succulent and looser in texture, the mucous membrane thicker, and shows a dark-red bleeding surface, while the parenchyma is often spotted with red from minute ecchymoses. The uterine glands of the fundus and body of the organ become lengthened, corresponding with the increase in thickness of the mucous membrane, so that the latter appears quite fibrous on section. The ciliated epithelium of the mucous membrane falls off at the beginning of the menstrual hæmorrhage, and a proliferation of epithelial cells, which resemble those of the Malpighian layer, begins. This proliferation continues through the whole of the menstrual period, till at its close the formation of ciliated epithelium begins again. The mucous membrane of the cervix is little altered. The two lips of the vaginal portion become of equal length.

MALPOSITIONS OF THE UTERUS.

No organ of the body is liable to so frequent and so varied and extensive changes of position as the uterus, many of which very materially affect the health of the individual and her prospects of maternity. Opinions are much divided as to the importance of some of these lesions; and it is uncertain how far the symptoms sometimes attributed to them are due to this cause alone; but into these questions we cannot enter. There are two great classes of malpositions, known respectively as versions and flexions.

Versions are those in which the whole organ is tilted out of its ordinary position, its axis being little, if at all bent on itself; and *flexions*, in which the axis of the cavity of the uterus forms an angle, more or less abrupt, with the cervix. We have thus anteversion and retroversion, and anteflexion and retroflexion, besides the occasional oblique position to one side, encountered in pregnancy, or associated with growth in the uterus. Of the two chief abnormal positions, the backward tilt and twist (often conjoined) are far more frequently noted than displacements forward.

It is obvious that extreme flexion of the uterus on itself, besides interfering gravely with the bladder or rectum, may be the cause of many painful symptoms and severe dysmennorrhœa. In Dr. Graily Hewitt's work on "Diseases of Women" (3rd Edition, 1872), the other symptoms thought to be associated with the less marked degrees of displacement are recorded at length, and the volume may be advantageously consulted for further information on the subject.

The second class of malpositions consists in a descent of the womb into the vagina, or in its extrusion beyond the labia; the term prolapsus has been, somewhat arbitrarily, applied to the lower degree, procidentia to the extreme form. In either case the axis of the womb must be altered, as well as its relations to the surrounding viscera. The predisposing cause is often some hypertrophy of the organ, together with a lax state of the tissues generally, and more particularly of the ligaments of the uterus and of the vagina, which may be the symptom of a debilitated constitution, as in lymphatic individuals, or the result of repeated pregnancies, the commencing prolapse being often materially aggravated by long standing; hence the frequency of this affection amongst cooks and laundresses. The immediate cause is very frequently an unusual bodily effort.

It is to be borne in mind, however, that in the majority of cases of so-called prolapsus the condition really present is an elongation and hypertrophy of the cervical portion of the uterus. This has been specially insisted upon by Huguier, who has also drawn attention to the frequency with which the portion of the cervix situate above the vagina becomes affected. This hypertrophy and elongation may be so extreme as to give rise to a very large tumour, dragging down with it both bladder and rectum and protruding from the vulva, the body and fundus of the uterus retaining their normal size and position. The secondary effect upon the prolapsed organ is, that it is irritated, and that its surface ulcerates; or that it becomes the seat of further congestion and hypertrophy, and that its exposed surface becomes indurated and horny. Prolapsus is most frequently met with after the middle period of life; instances of its occurrence before puberty are recorded by Dr. Ashwell,* and other authors. Dr. Ashwell's work also contains the history of three cases in which, during the whole period of pregnancy, the womb had lain partly or entirely external to the pudenda. In two of these the child was born while the entire uterus was beyond the vulva; in one it had occupied that position for several months, in the other for eight years previous to conception.

A very serious malposition, which comes on after parturition, spontaneously, or as the result of undue manual interference in removing the afterbirth, in an unusually distended or relaxed womb, is that known as inversion of the uterus. It consists in a greater or less descent of the fundus uteri into the cavity of the organ, and it may amount to a complete turning inside out. It is generally accompanied by very dangerous hæmorrhage; if the organ is not at once replaced in its proper position, and the patient survives the immediate shock; as sometimes happens, the uterus becomes reduced in size, and the inconvenience sustained may be comparatively trifling. Burns details a case in which an inverted uterus was borne for twenty years, menstruation continuing during the whole period.

Inversion is not, however, exclusively a sequel of parturition; it also occurs as a result of the influence of fibrous polypi, growing from the inner surface of the fundus. An unimpregnated inverted uterus is preserved in the Museum of the Royal College of Surgeons of England (No. 2,654), showing the Fallopian tubes obliquely in the upper part of the vagina—the effect of a polypus growing from the fundus. Velpeau removed a polypus from a woman, who died soon after of peritonitis, and the uterus was found to have been completely inverted. The presence of fibrous tumours in the substance of the uterus, or inclosed in the cavity, when complicating pregnancy, favours the occurrence of inversion by disturbing the normal balance of the expulsive contractions. Some authors are inclined to attribute it to extreme shortness of the umbilical cord. Instances are recorded in which the entire inverted uterus has been removed by ligature, or by the knife; in some cases inadvertently, owing to the tumour having been mistaken for a polypus, and of the patient's having entirely recovered. One of these cases is recorded by Dr. J. Cooke.*

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* "A Practical Treatise on the Diseases peculiar to Women," p. 541. London, 1845.

The diagnosis of inversion of the uterus from a large projecting polypus is not so easy as might be thought *à priori*. The chief reliable sign is the possibility of insinuating the uterine sound beyond the neck of the polypus into the cavity beyond. Of course no such sulcus exists in cases of complete inversion of the uterus.

We have already had occasion to refer to the occurrence of rupture of the uterus as a concomitant of pregnancy in the horned or bilocular malformation of the organ. The accident is also met with in the normal uterus. A trifling laceration at the os tincæ occurs at every birth, and is therefore of no consequence; and it appears that until the solution of continuity extends beyond the circular fibres of the cervix no danger is to be apprehended. Above this point the rupture may penetrate the entire thickness of the parietes, so as to allow an escape of the fœtus into the abdominal cavity; or one layer only, either on the inner or outer surface, may give way. It has been shown by many observers that the peritoneal investment of the uterus may, during parturition, alone be lacerated, leaving the uterine substance entire. The direction of the rent is stated differently by authors. Rokitansky affirms that it is generally vertical, Burns asserts it to be transverse, and Kilian maintains that it is commonly diagonal. It very rarely affects the fundus, but most frequently the posterior and inferior surface, which corresponds to the promontory against which, in the act of parturition, the expulsive efforts propel the child with peculiar force.

Rupture of the uterus occasionally takes place before parturition, as a result of external injury; it is said not to be necessarily fatal, nor as dangerous as might be supposed. In one such case of rupture in the fourth month of pregnancy, with no clear history of external injury, the parts were sent up from Norfolk to us for examination. We found a granular fatty condition of the uterine tissue at the site of rupture, suggesting some inflammatory process which had weakened the muscular wall at this spot and favoured its rupture, possibly by some fœtal movement, or by some comparatively slight external injury. Its occurrence during parturition is, unfortunately, not a mere pathological curiosity. According to the statistics of the Dublin accoucheurs, Drs. Collins and Clarke, the average frequency is about one in five or six hundred births. The former met with thirty-four cases in 16,414 births, the latter had four cases in 2,484 parturient females.

INFLAMMATION OF THE UTERUS.

Under this head we have to consider inflammation of the lining membrane, of the muscular walls, and of the surrounding structures. Independently of the puerperal state, it is at the period of menstruation that the uterus is most liable to all forms of inflammation. We shall first describe separately the non-puerperal forms.

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* "On the Removal of the Uterus in cases of Prolapsus and Inversion." London, 1836.

Endometritis, or inflammation of the lining membrane, is the commonest form. It may be acute or chronic.

Acute endometritis, or uterine catarrh, affects more especially the body and fundus of the uterus, the cervical portion being little altered. The mucous membrane appears deeply injected, swollen, velvety, and sometimes partly loosened. The glandular orifices appear prominent and open, the gland tubes themselves elongated. Minute hæmorrhagic spots are sometimes seen. The vaginal portion of the uterus is also injected and swollen. The secretion is at first clear, afterwards purulent. The inflammation may spread to the Fallopian tubes or to the vagina, if it has not originated there. It often passes into a chronic form.

Chronic endometritis, or catarrh, is a part of the affection commonly known as leucorrhœa. It affects more especially the cervical portion, which does not appear injected, but shows an unusual number of ovula Nabothi, which are also enlarged and often become pedunculated, hanging down as minute polypi. The vaginal portion is enlarged, spongy, and shows a characteristic red granular surface. Ulceration of the cervix and os uteri often result from long-continued catarrh, and present an eaten, corroded appearance. Obstruction of the os internum or externum may also result. The discharge is either viscid, straw-coloured, and transparent, or else red, purulent, and even sanguinolent.

Metritis, or inflammation of the substance of the uterus, independent of the puerperal state, is very rare. It may follow injury or irritation, or may result from the presence of new growths, or from other sources. The uterus swells, becomes softened, reddened, unusually succulent, and infiltrated with small extravasations. There is always at the same time endometritis, the mucous membrane being swollen and softened. This acute metritis may spread to the peritoneal cavity of the uterus and of the neighbouring organs. In rare cases it is said that suppuration occurs, and abscesses are formed in the uterine walls. On the other hand, the affection may result in complete resolution, or may pass into chronic inflammation.

Chronic metritis, according to Scanzoni, occurs in two stages—(1) a stage of infiltration, or softening, with hyperæmia; (2) a stage of thickening, or induration, accompanied by anæmia. The chronic inflammation results from chronic congestion, induced by heart disease as well as from acute disease. It is always accompanied by chronic catarrh.

Perimetritis is the name given to inflammation of the peritoneal covering of the uterus, which, as usually involving the neighbouring parts, may also be called pelvic peritonitis. It gives rise to the adhesions of the uterine and adjacent organs so often seen at post-mortem examinations.

Parametritis is used to signify inflammation by the side of the uterus—that is to say, of the subperitoneal connective tissue, and corresponds to the older term pelvic cellulitis. It may lead to abscess or to simple chronic induration. It is chiefly important in post-puerperal conditions.

Croupous, or Diphtheritic Inflammation.—In certain conditions false membranes, such as are seen in croup and diphtheria, are found on the uterine mucous membrane, chiefly as a secondary affection, in cases of typhus, cholera, the exanthemata, usually with similar affections of the vagina.

Dysmenorrhœa Membranacea.—False membranes forming a complete cast of the organ are sometimes thrown off, which must be carefully distinguished from those just mentioned. They are, in the words of Dr. Barnes, "three-cornered bags, somewhat longer in one direction, having an irregular opening at each angle. The opening at the smaller end, which is wider than the others, corresponding to the *os internum*, the others with the ostia of the Fallopian tubes." The membranes are rough ; on the outer surface they show ragged projections, corresponding to the uterine glands, the orifices of which are seen on the smooth inner surface. The membrane itself is composed of the epithelium, or nearly the whole mucous membrane of the uterus.

These casts are thrown off during the menstrual period, with symptoms of dysmenorrhœa ; hence often accompanied by hæmorrhage. From its precise resemblance to the decidua of pregnancy, the membrane may be called a "menstrual decidua." According to Dr. Barnes, a simply fibrinous membrane is sometimes expelled under the same circumstances. Some good examples of dysmenorrhœal casts are in the Museum of St. Thomas' Hospital.

Ulceration.—Dr. T. Smith states that ulcers of the os uteri may be the primary result of inflammatory action, or arise from eruptive disorders of the mucous membrane, similar to herpes or eczema of the skin. But they more frequently result from the chronic irritation produced by the discharge from the cervix. This is confirmed by the fact that, except in eruptive disease, the os uteri is rarely found abraded, unless there is co-existent disease of the glandular portion of the cervix. Tubercular or scrofulous ulceration produces deeper-seated destruction of its tissues ; it gives rise to more profound ragged erosion on the surface and sides of the os, which, however, is not accompanied by that knotted induration which is generally characteristic of the carcinomatous ulcer. Nor is it marked by the same tendency to spread and involve adjoining parts in its destruction.

In addition to the forms of ulceration already spoken of the uterus is subject to the specific ulcers of primary and secondary syphilis. Under the name of corroding ulcer, Dr. John Clarke has described a variety of destruction which differs from cancer only in not being accompanied by an indurated deposit. No account of the microscopic appearances of the part so affected is on record; a link is therefore wanting to enable us to pronounce positively as to the nature of the disease. It is probably a form of "rodent ulcer," called by the late Mr. C. Moore "rodent cancer," and having marked affinities to epithelioma. The absence of induration, the cleanly cut margins of the sore, the want of fixation of the uterus, and the comparatively slow course of the disease, all tend to separate it pretty widely from the ordinary forms of cancerous ulceration. It is of very rare occurrence; Dr. Ashwell, in the course of an extensive practice of twenty years, only twice met with it.

ACCUMULATIONS IN THE UTERINE CAVITY.

Hydrometra.—The cavity of the uterus may become dilated by an accumulation of fluid within it. This occurs, independently of the accumulation of menstrual blood, in individuals past the climacteric, in whom some catarrhal or other inflammatory condition exists, and is combined with obstruction of the orifice, usually of the os internum. In such cases mucus or pus may accumulate, and distend the uterus to a considerable size—not often larger than the fist—very rarely to the size of the pregnant uterus at full time. The walls are usually thin, and more fibrous than muscular; the mucous membrane atrophied and pale. The accumulated fluid undergoes, in time, considerable changes, and though it may have been at first purulent or viscid, it becomes in the end watery and thin, sometimes colourless, sometimes yellow or brown. If the os externum be obstructed, both the body and cervical portion become dilated; either into one cavity, or if the os externum be also narrow, into an hour-glass shape. The obstruction of the orifices giving rise to this condition may be caused by inflammation, by cystic polypi of the cervix, by flexion, or by other causes.

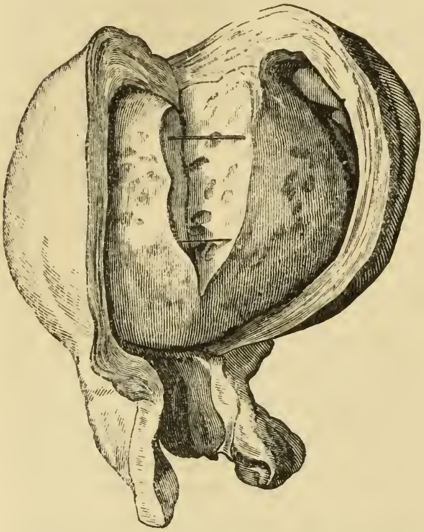
Hœmatometra is the name given to accumulation of menstrual blood in the uterus, through congenital closure of the hymen, the external parts, or, more rarely, of the os externum or internum. The dilatation may be, especially in cases of imperforate hymen, very great indeed, at least equal to that of pregnancy. The relaxed blood becomes thick, black, and tarry.

Physometra, or accumulation of air in the uterus, is sometimes observed after severe labours, as the result of decomposition of placental residues.

MORBID GROWTHS—FIBRO-MUSCULAR TUMOURS AND POLYPI.

The abnormal formations that most frequently present themselves in the uterus are fibroid or fibro-muscular tumours; they occur imbedded in the texture of the organ, or protruding from its inner surface in the cavity, or from some part of the external surface. When projecting into the cavity of the uterus they receive the name of fibrous polypi. While imbedded in the uterine tissue they form globular, white, glistening, dense, tumours; there may be only one, or they may be numerous. In preparation, No. 2,674, in the Royal College of Surgeons, we see a uterus, with from eight to nine large fibroid tumours in its walls, varying from one to four inches in diameter. In size they differ even more than in number; they are seen in every gradation, from that of a pin's head to that of a melon. The preparation spoken of further on is an instance of the great development they attain. Dr. Lee mentions one weighing fifty-four pounds, which contained several cysts filled with fluid. The fibroid tumour is surrounded by a membrane which separates it from the uterine tissue, so that there is no very intimate union between the two structures. "The most usual position for these tumours," according to Mr. Lee's analysis of seventy-four cases, "is the submucous, viz., those projecting into the cavity of the womb, and the pedicles of these are generally situated just below the openings of the Fallopian tubes. The next position in which they are most abundant is the posterior wall and fundus of the uterus; they are very rarely situated in the anterior wall, and still more rarely in the cervix uteri." Of the general truthfulness of these remarks, every one may convince himself by glancing through one of the metropolitan museums of pathological anatomy. It appears that the nearer the original growth takes place to the mucous surface of the uterus, the more a gradual extension of the entire tumour into its cavity is likely to ensue. In this way we account for the gradual elongation of the pedicle, which after a time is the only connection between the tumour and its matrix; it may then be removed with comparative facility by operative procedure. The pedicle is not, however, a necessary consequence of the arrival of the fibroid tumour at the external surfaces. In one of the largest specimens which has come under our notice, (Royal College of Surgeons; No. 2,672) which is also remarkable as affecting the cervix exclusively, the remainder of the

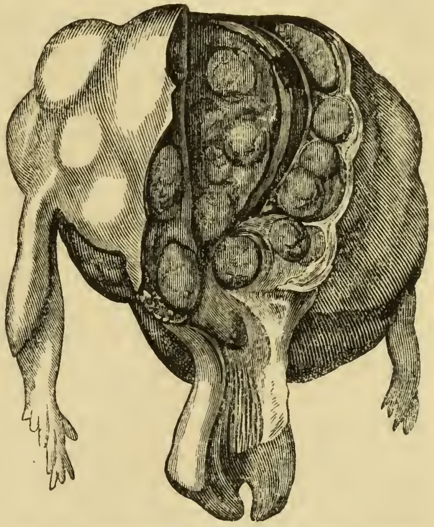
Fig. 179.



Fibro-muscular tumour projecting into the cavity of the uterus.
(St. George's Museum, 128.)

uterus continuing normal, we find no attempt at the formation of a pedicle; the tumour has evidently formed in the substance of the posterior part of the cervix, and in its growth has separated the uterine tissue, which is still spread out over the upper part of the tumour as if embracing it. The tumour, in this case, is twelve inches long, by five inches thick, and presents the ordinary structure of fibrous tumours. The fibroid tumours found almost free in the abdominal cavity, or, at least, only attached to the uterus by cellular adhesions, probably have the same origin as the growths we have just considered; having been developed in the first instance under the peritoneal investment of the uterus, they have subsequently become detached. This does not preclude the possibility of their being formed primarily, at the points where they are found.

FIG. 180.



A uterus, the upper half of which is enlarged by the growth of numerous fibromuscular tumours in its walls. One tumour, larger than the rest, projects into the dilated upper part of the cavity of the uterus, and completely fills it. Five others are shown by the section imbedded in the anterior wall, and many others project on the external surface of the uterus. The lower half of the uterus is healthy, but elongated. The walls of the portion occupied by the tumours are thick and laminated, like the walls of the uterus in pregnancy.

(St. Bartholomew's Museum, xxxii. 16.)

Minute Structure.—The intimate structure of fibroid tumours varies in some respects; to the naked eye it exhibits, at times, a concentric disposition of fibres; but more commonly an irregular wavy appearance, without any uniformity of arrangement, presents itself, and it is more particularly in this case that cavities containing blood, a dark-coloured gelatinous fluid, or a clear serum, are formed, which give the tumour, on section, a resemblance to the sero-cystic disease of the testes or mamma. Occasionally the fibrous tumour presents a lobulated conformation. The microscopic appearances of the fibroid tumours of the uterus are not in accordance with what we should expect to find in a true fibrous structure. The microscope, in fact, demonstrates that they belong to an altogether different class of growths; the fibrous appearance is scarcely perceptible under the microscope, which displays elongated nuclei, imbedded in an amorphous stroma. It appears to us that from the analogy they present to the genuine uterine tissue, in the unimpregnated state, we should be more disposed to class them with homologous than heterologous productions; that they should be regarded rather in a relation to the womb analogous to that of exostosis to the matrix it springs from, than of a character totally at variance with that of their nidus.*

[The foregoing paragraph, with its accompanying note, have been left as they stood in the edition of this work published in 1854. It may be added here, that the views then expressed have been since abundantly confirmed by subsequent observers. Indeed, thin sections of these tumours, stained with carmine, show so distinctly the structure of bands of long spindle cells of plain muscular tissue, with rod-shaped nuclei, crossing and recrossing, as in the uterus itself, that the name *myoma* is now given to these growths instead of the older term fibroid, which expressed less definitely their muscular nature.—Ed.]

The amount of blood supplied to these muscular tumours varies. The majority are but scantily provided with vessels. Some, when injected, only exhibit one or two larger vessels traversing the substance of the mass; others exhibit considerable and uniform capillary injection, the vessels being distributed in the connective tissue. The hæmorrhage to which fibroid growths of the uterus may give rise is not owing to a laceration of these vessels, but to the irritation and congestion they induce in the superincumbent mucous membrane, which, from the same cause, may ulcerate and slough. When complicating pregnancy, they induce hæmorrhage, by preventing the normal development of the organ; hence they are very apt to give rise to miscarriages. Fortunately for the individuals, they are often a cause of barrenness.

These tumours are usually of very slow growth, sometimes in the course of ten or twelve years reaching the size of a melon, and in rare instances attaining enormous dimensions. Specimens are on record weighing forty-four and even seventy-one pounds, and blocking with their great bulk a considerable portion of the abdomen. They often, however, remain quiescent for many years, and they are prone to undergo the degenerative changes common to all morbid growths. Thus they may be the seat of extensive fatty degeneration, or of calcareous infiltration, or of mucoid softening into irregular cysts, forming the fibro-cystic tumour of the uterus. An excellent example of one of these tumours which has undergone calcareous degeneration to a very marked degree is preserved in the Museum of the Middlesex Hospital. It was removed by Mr. J. Moncrieff Arnott† from the body of an old lady, who, having died at the age of seventy-two, from the effects of a fall, was found to have a tumour, weighing five pounds, and as hard as marble, in the uterine parietes, which had become converted into a mere membrane. The tumour had been diagnosed as scirrhus when she was at the age of forty. It was found on analysis to contain nearly two-thirds of phosphate of lime.

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* Since the above was written, a corroboration of the view expressed has been published in the "Report of the Pathological Society for 1853," p. 219. Dr. Bristowe, in an elaborate paper on the subject of fibrous tumours of the uterus, concludes, from his examinations of them in the impregnated and unimpregnated conditions, that all so-called fibrous tumours of the uterus—at least in their earlier stages, before degeneration has taken place in them—are essentially muscular tumours; not simply fibrous tumours with a greater or less quantity of muscular fibre mixed up with them, but developments of true and undoubted muscular tissue.

† "Med.-Chir. Trans." vol. xxxiii. p. 199.

Clinically it is found convenient to distinguish certain varieties of muscular tumours of the uterus according to the precise part of the uterine wall in which they appear. Thus, those on the outer surface—*sub-peritoneal;* those lying imbedded in the thicknes sof the wall—*parietal;* those projecting somewhat into the cavity—*submucous;* and the further stage of this last, when the growth swings by a pedicle into the uterine cavity—*fibroid polypus*, have all special habits of growth, and are attended with special symptoms. The first, sub-peritoneal, usually attain the largest size, and the last, polypi, interfere most gravely with the uterine function, giving rise to hæmorrhage, and occasionally being spontaneously expelled by the uterus, which hypertrophies for the purpose, as in pregnancy; whilst the parietal tumours not unfrequently play an important part in the production of the flexions noted on a previous page.

But, pathologically, all are alike overgrowths, more or less circumscribed, of the normal uterine tissue, and are subject to the same periodical change of bulk, at the same time having an independent tendency to continuous growth, and a liability to the various degenerative processes common to all new formations. So perfect is the muscular structure of these tumours before degeneration has set in, that they are capable of spontaneous contraction and relaxation, as has been observed by Virchow and others.

Dr. Barnes has described (St. Thomas's Hospital Reports, 1871) a form of polypus under the name of hypertrophic polypus. This growth is a pedunculated mass of plain muscular tissue, usually of small size, and often multiple, springing from within the greatly hypertrophied neck of the so-called "prolapsed" uterus, and causing the hæmorrhage which not unfrequently complicates these cases.

Fibro-muscular tumours have not been observed before puberty. Dr. Lee agrees with the statements made by Bayle, that they are most frequent in virgins, and that they exist in twenty out of a hundred middle-aged women.

MUCOUS AND GLANDULAR POLYPI.

Besides the pendulous myomata, or fibro-muscular growths, which are the commonest form of uterine polypi, there are certain soft, succulent growths which occasionally project into the cavity of the uterus or its mouth, either sessile or with long slender pedicles, and which are obviously overgrowths or outgrowths from the mucous lining of the organ, and very different from the solid fleshy tumours just described.

These softer—and, for the most part, much smaller—masses correspond to the jelly-like polypi met with in the nose, and consist almost wholly of altered mucous membrane. Hence they may present the characters of myxoma, the sub-mucous connective being actively developed, and its interstices filled with clear viscid fluid, the whole being invested with a delicate epithelial layer; or, the glandular portion of the mucous membrane being the special seat of the hypertrophic change, a soft vascular tumour is formed whose structure is made up almost entirely of gland tissue, ducts, and their terminal dilatations, regularly lined with epithelium, and connected by more or less delicate bands of fine fibrous tissue, in which the vessels are distributed. To this last class belongs the "channelled polypus" of Dr. Oldham, described as made up of several long channels, with occasional communications between them, and opening by large orifices on the free surface of the growth.

A third variety is formed by the expansion of the glandular crypts after obstruction of their ducts, and these tumours are more distinctly vesicular or cystic, the tiny spheres being distended with the accumulated mucous or other secretion, seldom attaining any large size, and as rarely becoming pedunculated. The "ovula Nabothi," or firm clear vesicles in the os and cervix uteri, belong to this class.

The symptoms caused by the presence of these succulent polypi are of course far less important than those attending the larger fleshy growths; they can hardly interfere with parturition, but the hæmorrhage to which they frequently give rise may render operative interference necessary for their removal.

UTERINE CYSTS.

Cystic growths are extremely rare in the uterus. Certain cases are on record, however, in which the cystic transformation already mentioned, affecting muscular tumours of the uterus, proceeds to such an extent as to make the cyst development the leading feature of the case, and such tumours have derived a grave importance of late from their close simulation of ovarian dropsy. In almost all the instances of attempted removal of the tumour under this supposition, death has resulted. An interesting example of this cystic tumour of the uterus was brought to the Pathological Society of London in 1867 by Mr. Isaac B. Brown. The tumour in this case—which is sufficiently typical for brief narration here—weighed eight pounds and a half after some pints of thin, clear fluid had been removed from an enormous cyst in its substance, and it sprang from the body and neck of the uterus, having been evolved out of the substance of these parts. The wall of the cavity, and a large fleshy projection into its midst, were made up for the most part of fibro-muscular tissue, more or less closely resembling the tissue of the uterus itself; but, together with this, forming a structure resembling the pancreas, was a certain amount of cell-growth, grouped in small masses, and suggesting glandular tissue. Many irregular cysts communicated freely in all directions, giving rise to the large cavity already mentioned, which contained upwards of four pints of fluid, and these cysts appeared to have been formed by the accumulation of fluid in the spaces of the loose fibroid structure.

The diagnosis of these rare growths is necessarily beset with the greatest difficulty, the chief points of diagnostic importance being the slow growth of the tumour. When a preliminary incision has once been made through the abdominal wall, the dark colour of the presenting tumour, and the thin, often blood-stained, fluid escaping through the trocar, instead of the usual viscid ovarian fluid, are mentioned by Spencer Wells as additional evidence of the supposed ovarian cyst being really uterine.

True hydatid tumour of the uterus is extremely rare, although occasionally small hydatid cysts with their characteristic structure have passed *per vaginam* from an intra-abdominal growth.

Hydro-metra and *hæmato-metra*, or collections of fluid or blood within the dilated uterus, are now and then met with, associated with some form of occlusion of the uterine mouth, or with the graver varieties of flexion; but such cases would be readily distinguished from fibro-cystic tumours.

TUBERCLE OF THE UTERUS.

Tubercular disease in the uterus affects chiefly the lining membrane, where we find the whole mucous surface covered with a layer of soft cheesy, or white putty-like material, sometimes almost puriform, and even in sufficient quantity moderately to distend the uterine cavity. In this state it is often very difficult to demonstrate the presence of tubercles or of anything beyond degenerative inflammation of the mucous membrane; and in the cases which have fallen under the observation of the editor no tubercles were seen in the uterus, though in some cases present on the serous surface. In other cases, however, they may be present on the mucous membrane, either before the degenerative process has gone so far, or under the layer of degenerated caseous material. In early stages of the disease the surface is described as covered with grey miliary tubercles. Under the caseous mass, in advanced cases, the uterine substance is found infiltrated and partially destroyed. Other parts of the organ show hypertrophy, whether fibrous or muscular we cannot say, but probably the former. The disease especially affects the fundus, and is almost always bounded by the *os internum*. Destruction of the wall and perforation into the peritoneum may occur, but are rare.

The uterine disease is most usually associated with a similar affection of the Fallopian tubes, and sometimes spreads from these. Very rarely it extends to the cervix uteri and vagina. Almost always there is tubercular disease of other organs also, but in some cases not. The affection is comparatively rare. Louis found three cases out of 200 phthisical women.

Syphilitic tumours have been in one case seen in the substance of the vaginal portion of the uterus; syphilitic ulcers or chancres are more commonly seen there, and sometimes syphilitic condylomata or papillary growths.

Lardaceous degeneration of the uterus has been observed. The organ was enlarged, pale, and translucent.

CANCER OF THE UTERUS.

Cancer of the uterus is a disease of frequent occurrence. Dr. Lever* has shown that the proportion of cancer to other uterine affections is as one to seven, or about thirteen per cent. The period of life most obnoxious to it is that between the fortieth and fiftieth years; and though numerous examples are met with earlier in life, the statement of Boivin and Dugès, that in 409 cases they found twelve under twenty years of age, can scarcely be credited, unless, as we are assured by Dr. Walshe, uterine cancer is more prevalent in the French than in our own capital. The analysis of their cases yields the following table:—

Under 20 years of age . . .	12 cases.
Between 20 and 30	83 ,,
,, 34 ,, 40	102 ,,
,, 40 ,, 50	201 ,,
,, 50 ,, 71	11 ,,

They attribute a great share in the causation to sexual excesses, but this view is now generally given up. Celibacy does not appear to favour its development; the ratio, according to Dr. Lever's analysis, is: single women, 5·83 per cent.; widows, 7·5 per cent.; and married females, 86·6 per cent. This is found to be identical with the relative frequency of other uterine affections in their respective classes. The increased activity of the uterus and its frequent changes of nutrition, occasioned by multiple parturitions, seem to exercise a definite influence on the production of cancer, for Mr. Sibley's and Dr. Tanner's statistics both show an excess above the average in the number of children borne by women afterwards the subjects of uterine cancer. In the one set of cases this increase amounted to thirty per cent. over the average fruitfulness of all marriages, and in the other set the families averaged two and a half children more than usual.

As a rule, the cervix or os uteri is the part first affected, a feature which broadly distinguishes this disease from fibroid growths. A very remarkable exception is presented in a specimen of cancer of the body and fundus uteri in St. George's Hospital Museum, in which the cervix is entirely free from disease (No. 134). Irregular thickening and induration of the os and cervix with deep fissures may be the result of chronic inflammation and laceration, and as this is a curable affection, it is important to distinguish it. It is to be noted that in this form of deformity of the os uteri, the fissures radiate around the os, the lobes so formed being quite smooth, free from nodulation and from ulceration. On the other hand, in early stages of malignant disease, the mucous membrane of the os and cervix is studded with irregular hard tubercles, or a single knotty growth is present, the covering membrane being of a livid blue or crimson colour, and glued to the parts beneath. The occurrence of a roughly excavated ulcer on a hard base, and accompanied with much surrounding induration, and some fixation of the uterus, renders the more dreaded diagnosis pretty certain. The advance of the growth and the concomitant subjective symptoms, the fusion and ulceration, the implication of the surrounding parts in the process, the fixation of the womb, and the rigid nodulated degeneration of the vaginal mucous membrane, together with sanguineous fœtid discharge, soon enable us to form a positive opinion if the disease be malignant.

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* "Med.-Chir. Trans." vol. xxii. p. 267.

The disease spreads more or less rapidly to the adjoining parts; to the vagina, the rectum, and the other pelvic viscera, the whole being matted together in extreme cases, and presenting a frightful spectacle of disorganization and cancerous destruction. The ulceration that leads to this result, while it gives rise to fœtid vaginal discharges, causes very extensive loss of substance of the parts first involved; the vaginal portion of the uterus, and the vagina itself, are the first to be eroded, and gradually communications are established between the various abdominal organs; the destructive character of the affection nowhere manifesting itself with the virulence that it here exhibits. All this havoc is what we might reasonably expect to ensue from the presence of any morbid infiltration leading to rapid cell growth and early decay in a free mucous surface abundantly supplied by vessels and in constant friction against an opposed mucous surface. No matter what the special anatomical cell-growth, such irritation would be certain to set up inflammatory processes in its immediate neighbourhood, and the free secretions of both uterus and vagina tainted by so grave an irritant would speedily cause the spread of such malignant destruction in the manner commonly witnessed.

This uniformity of the destructive process in growths widely differing in their anatomical structure, when attacking the os and cervix uteri, has been pointed out by Mr. Henry Arnott,* in a review of 136 cases of uterine cancer noted by him in the Middlesex Hospital, in fifty-seven of which he confirmed the diagnosis by post-mortem examination. The same writer has drawn attention to the wide discrepancies met with in the accounts by various authors of the form of cancer usually present in the uterus. Quoting several distinguished authorities he shows how contradictory are their statements on this head. Lebert, for example, finding epithelioma in only six out of forty-five cases, whilst Förster—an equally eminent observer—discovered forty-two instances of epithelioma in fifty-two cases of uterine cancer.

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* "Cases illustrating Certain Points in the Pathology of Cancer of the Uterus," by Henry Arnott, "Trans. Path. Soc.," vol. xxi. p. 281, et seq.

The form of cancer most often encountered in the uterus is probably carcinoma, such as commonly attacks the female breast, but the alveolar stroma is rarely well developed in this position. Far oftener the firm white infiltration is found to be made up of densely aggregated cells of very various sizes and shapes, grouped into irregular clusters by an open, delicate fibrous stroma, or infiltrated amongst the uterine tissues. Epithelioma is, however, very frequently met with—not, indeed, in the characteristic form of "cauliflower excrescence," which is a comparatively rare phenomena, but occurring as a ragged ulceration, whose white friable base is found to consist largely of squamous (in rare instances columnar) epithelial cells, rapidly proliferating in the deeper uterine tissue, and occasionally even presenting the well-known "bird's-nest" bodies.

Sarcoma, either spindle or round-cell, is also liable to attack the uterus, and to affect the cervix, and spread thence just as do the other forms of malignant cell-growth. According to Gusserow, sarcoma is more apt to invade the fundus than the cervix, and he states, indeed, that it alone does attack this portion of the organ. We have ourselves, however, seen two cases of spindle-cell sarcoma commencing in the cervix and running the usual course; whilst we have more than once found detached nodules of carcinoma lying in the uterine wall at a distance from this position; and we once examined the body of a woman whose uterus was wholly destroyed by distinct carcinoma, excepting the os, which was quite free. Gusserow also states that the soft infiltrating growth met with at rare intervals attacking the mucous membrane lining the uterine cavity, the os remaining free, is a variety of round-cell sarcoma. According to this observer, pain is a symptom complained of much earlier in sarcoma than in carcinoma or epithelioma of the uterus, from the stretching of the uterine tissues around the imbedded nodule, and the consequent lesion of the fine nervous filaments. Some authors describe mixed forms of sarcoma and carcinoma.

Owing to the very rapid destruction which always accompanies malignant disease of the uterus, the disintegrating cancer material crumbling away in gangrenous sheds and melting into the fœtid vaginal discharge, and to the almost constant inflammatory changes which run a simultaneous course, it is often extremely difficult to make out the minute structure of the disease. Not seldom the sloughing action spreads beyond the line of new growth, and after death considerable portions of vagina and uterus are found softened, green, and sloughing.

The tendency to affection of the lymphatic glands, and to the occurrence of secondary growths in remote parts of the body, has for the most part escaped the detailed notice of writers on the subject. In Mr. Arnott's fifty-seven cases of autopsies of uterine cancer already referred to, the lymphatic glands were involved in twenty cases, and in eleven the viscera contained secondary growths. His observations seem to show that these multiple foci of disease are far more common in carcinoma and sarcoma, although in the later stages of epithelioma the glands may occasionally suffer.

Besides these ordinary varieties of uterine cancer, papilloma of the mucous membrane of the os and cervix may be met with, the normal papillæ becoming greatly hypertrophied with thickened layers of investing epithelium, but without any infiltration of the epithelial elements amongst the deeper tissues.

We have seen also a general villous condition of the lining mucous membrane forming soft vegetations projecting into the cavity of the uterus, and showing under the microscope branching papillæ of connective tissue clothed with columnar and sometimes exfoliated epithelium.

Although uterine cancer is commonly primary, secondary nodules, both of sarcoma and carcinoma, may spring up in the uterus, the primary tumour being in other and remote parts of the body. In these cases the nodules are usually imbedded in the fundus or project from its peritoneal surface. We have seen also an instance of well-marked colloid cancer invading the fundus of the uterus from an adjacent wide-spread colloid of the peritoneum.

CHAPTER XLII.

ABNORMAL CONDITIONS OF THE OVARIES AND MAMMÆ.

THE OVARIES.

Congenital Malformations.—No instance is known of excess in number of the ovaries, but they are sometimes congenitally large, and then show an excess of fibrous tissue. They are hardly ever found deficient, except when the uterus is wanting also, but sometimes when this is only rudimentary. One ovary is sometimes wanting when a bicornute uterus is deficient on the same side. Rudimentary development of the organs is indicated by the want of follicles or their rudiments. Their condition is usually accompanied by the want of sexual character, and by imperfect development of the other generative organs. The imperfection is sometimes unilateral.

Displacement of the ovaries is seen in ovarial hernia, where the gland lies in a pouch of intestine, outside the abdominal wall, like a hernial sac. Sometimes the pouch resembles the inguinal canal of the male, and we have an inguinal ovarial hernia. In Pott's celebrated case, a double hernia of this kind existed, and both ovaries were removed by operation, with the result, as is well known, of stopping menstruation and abolishing the female physical characters.

As the result of chronic or repeated inflammation, adhesions are often found by which the ovaries are fixed in abnormal situations; more especially are they found, tied down, behind the uterus.

Connection of the Ovaries with Menstruation.—The ovaries of children, who have never menstruated, are perfectly smooth. But after puberty, and when menstruation has occurred, accompanied by the rupture of Graafian vesicles, the surface becomes marked with puckered scars, surrounded by pigmentation. Hence, generally speaking, a perfectly smooth surface is evidence that menstruation has not taken place. Klob, however, quotes instances to show that scars may entirely disappear. In the ovaries of older persons the scars become very numerous, and the whole ovary acquires a wrinkled appearance; and there is at the same time wasting of the organ. This wasted and shrivelled appearance must not be regarded as morbid. If death take place during menstruation, the ovary is found turgid and vascular, injected on its surface; and showing the changes due to ovulation, now to be described.

Corpus Luteum.—It is necessary to be acquainted with the ordinary form of the scar left by a ruptured Graafian follicle, both when it is followed and when it is not followed by impregnation. In the latter case, when ovulation has just taken place, a cavity is found, some three-eighths of an inch in diameter, containing blood, fluid or coagulated. A variable quantity escapes through the rupture in the follicle, but some remains, and from this, with a growth from the follicular walls, the *corpus luteum* is formed. The blood goes through the changes already indicated, it becomes decolorized, probably softens into a pulp, and ultimately nothing may remain but some fibrous material, blood crystals, and pigment granules. The follicular wall simultaneously goes through changes of a proliferative kind, and forms a yellow or brownish zone, some half a line thick, often with a crenulated outline. This undergoes rapid fatty degeneration, and is ultimately absorbed with the remains of the blood, so that nothing remains but a whitish scar, with some pigmentation. This process of evolution may be nearly complete within the month.

If impregnation follow, the active flow of blood to the uterine organs causes the proliferative process to be much more energetic. A yellow zone, some three lines broad, is formed, so deeply folded or crenated as to have been compared to a leaf; within which is the altered blood clot, surrounded on all sides, except at the ruptured orifice. The whole may, at the maximum, measure eleven lines in diameter, and, even at the end of pregnancy, from three to six lines. The process of evolution is accordingly by no means complete, even after nine months.

Abnormal Evolution.—Sometimes a subsequent or secondary hæmorrhage takes place into the corpus luteum. Sometimes a sort of cyst is formed. Rokitansky has described the formation of the species of fibrous tumour from the scar, and in one case it has formed the starting point of cancer. A papillary growth from the corpus luteum, projecting as a dendritic protrusion through the rent in the follicle, has also been observed.

Hæmorrhage.—The normal escape of blood from ovulation may be excessive, and the Graafian follicle may be distended to the size of a nut, a hen's egg, or even more. If the excess of blood flow into the peritoneum, it produces one form of *Retrouterine Hæmatocele*, or accumulation of blood in Douglas's pouch; but this may be derived from other sources. Sometimes, in young girls, the hæmorrhage may produce laceration of the substance of the ovary, or ovarian apoplexy. Hæmorrhage may occur quite independently of menstruation, even in infants; there is, then, no discharge into a Graafian follicle. This condition has been observed in purpura hæmorrhagica and in some fevers.

INFLAMMATION OF THE OVARIES.

Many of the symptoms referred to inflammation of the ovaries during life are probably due to mere congestion, which produces hyperæsthesia. True inflammation, however, may occur, and this either in the puerperal state or independently of it. The former will be discussed hereafter.

Non-puerperal inflammation, or Oophoritis, may, according to Klob,* be either follicular or parenchymatous. In the former the follicles are swollen, filled with serous or purulent fluid and detached masses of the lining membrane (membrana granulosa); it leads to the destruction of the inflamed follicles, but no other important result. It is always confined to one ovary. In the parenchymatous form, which may be combined with that just mentioned, the stroma of the organ is affected, the ovary appears much swollen, vascular, and œdematous; and on section the follicles are scarcely visible. It leads to considerable destruction of the follicles, and may occur simultaneously on both sides. It is always accompanied by some inflammation of the peritoneal surface. This form of inflammation, especially if combined with the follicular form, may result in suppuration, though this is rare. More frequently it passes away, leaving behind it some induration and adhesion of the ovary to neighbouring parts, or may pass into a chronic form. The right ovary is far more frequently affected than the left (Klob).

CYSTIC DISEASE OF THE OVARIES.

The production of cysts in the ovary is a subject of great complexity and importance. On the one hand, a certain form of cyst-production is so common as to be of no practical importance, and almost to be regarded as normal; while, on the other hand, cystic growths may be among the largest and most formidable growths to which the body is liable. We must, accordingly, endeavour to classify the cases, that they may be conveniently discussed, under the following heads:—(1) Simple, or Unilocular Cysts; (2) Tubo-ovarian Cysts; (3) Compound, or Multilocular Cysts; and (4) Dermoid Cysts.

1. *Simple, or Unilocular Cysts.*—There is no doubt that these arise from enlargement of the Graafian follicles. It may not be super-

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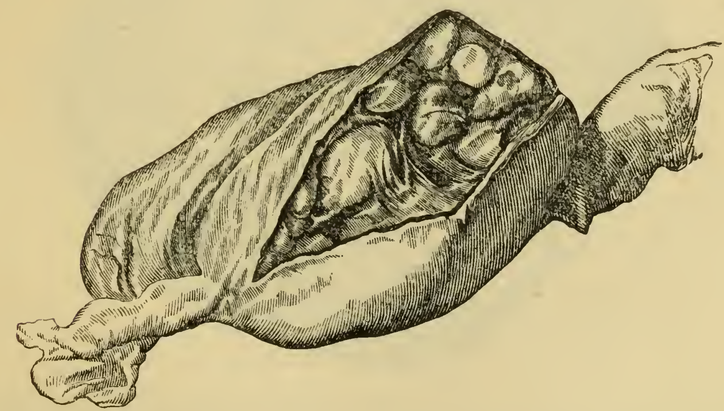
* "Die Pathologische Anatomie der Weiblichen Sexual Organe," Wien, 1864—a work to which we must specially acknowledge our obligations.

fluous to recall the fact that the Graafian follicle is itself a cyst, lined by a cylindrical epithelium, called the Membrana Granulosa, which is accumulated at one point into a granular mass (discus proligerus), containing the ovum. The follicle further contains a transparent albuminous fluid, called the liquor folliculi, the increase of which constitutes cystic enlargement. Waldeyer * believes this liquid to be continually added to by the disintegration of the follicular epithelium. It is worth noting, that the size of the mature follicle is ten to twelve millimetres = three-eighths of an inch in diameter. Simple cysts of this kind are met with in the fœtal ovary, and subsequently at all ages. As the dilatation increases the ovum perishes, but has been still demonstrated in cysts, the size of a bean or of a cherry, by Rokitansky and Ritchie. Several cysts are often found in the same ovary, but naturally the largest are those which occur singly. The larger cysts are lined by a simple pavement-epithelium. Their contents are either clear albuminous fluid, or the same variously coloured by the admixture of blood. The walls are, of course, much thicker than those of a normal follicle. Simple cysts are usually small, and rarely larger than a hen's egg; but instances as large as the fist, or still larger, are described. They often occur in both ovaries at once. The cause of their production is unknown, but they may occur quite independently of menstruation.

FIG. 181.



Incipient cyst formation. The ovary is represented of the normal size.

Another variety of simple cyst is that proceeding from a transformation of the corpus luteum, as first traced by Rokitansky. In such a case the wall of the cyst is lined by a layer of soft material, thicker than itself, and somewhat loosely attached, in

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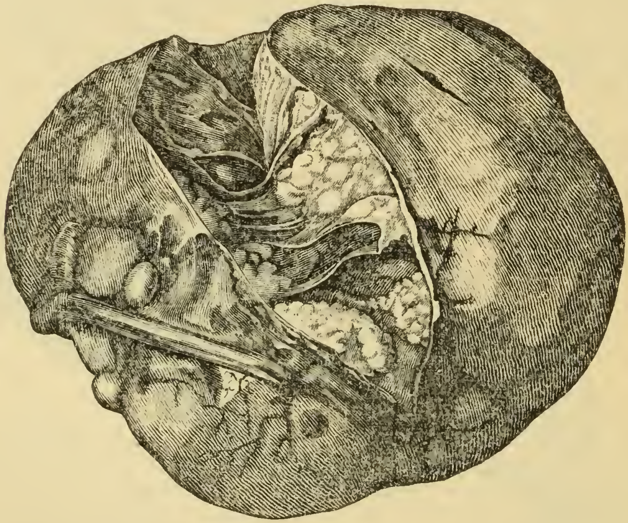
* In order to understand the pathological relations, reference should be made to Waldeyer's article on the ovary in Stricker's "Histology," vol. ii. p. 161, New. Syd. Soc. Translation, or to the same writer's large work "Eierstock und Ei." 1870.

which may be recognized the remains of the yellow portion of the corpus luteum.

2. *Tubo-Ovarian Cysts* are also developed from a follicle ruptured at menstruation, but from one to which the fimbriated extremity of the Fallopian tube has become adherent. The ovarial portion of the cyst is lined by a yellow or brownish zone, representing the corpus luteum, which becomes continuous with the wall of the tube. Only a portion of this, about the outer third usually, participates in the cyst-formation, and the enlargement is not considerable.

3. *Compound, or Multilocular Cysts; Cystoid Disease of the Ovary.*—The formation of these cysts is much more difficult to explain, and their relation to the simple cysts is uncertain. Before discussing the numerous theories which have been framed to account for them, we must endeavour to describe the various forms in which they occur. These are the structures which usually give rise to "ovarian dropsy," but similar symptoms may be produced by the simple cysts, if very large.

Fig. 182.



A multilocular ovarian cyst, removed from a female, aged twenty-nine, during life, by Mr. J. B. Brown. Septa form larger compartments, in which there is a secondary and tertiary growth of cysts. The tumour weighed 11 lbs. 3 oz.

The following account is chiefly taken from Klob:—

(*a.*) There may be one large, thick-walled cyst, in whose walls are contained smaller cysts, the shape of which is so modified by the presence of the large cavity, that they appear as if they had been formed by a splitting up of—or at least in the thickness of—the wall of the main cyst. In other cases we find a cyst, the inner wall of which shows partitions, which either project boldly into the cavity, or else are attached to the wall in the form of crescentic folds, or else are more or less flattened against the wall and adherent to it. It is plain that in such a specimen the large cavity has arisen from the confluence of several smaller ones, the walls of which have become reduced to incomplete partitions. These may again be reduced to mere bands crossing the cavity, and inserted into a part of the wall which, by its thickness, shows its original compound character.

(*b.*) A second form of compound cyst is that in which we find, on the inner wall of the larger cyst, an immense number of small cavities, sometimes arranged in groups, with very thin walls, containing either clear serous fluid, or else gelatinous, honey-like matter. A group of such smaller cysts sometimes projects from a single point in the inner wall of the larger, forming by absorption of the partitions a reticulated structure, containing pellucid or gelatinous fluid. Such a mass may, in great part, fill the larger cyst; and it is thought that, by completely filling it, the reticulated appearance of some large cysts is produced. Again, a single secondary cyst may, in like manner, grow out from the wall of the primary till it completely fills it. As this secondary cyst may be affected in the same way, of course great complexity may result. Papillary excrescences sometimes grow from the wall of the cyst, and project into the interior. The papillary terminations may be either solid, or themselves cystic. These growths may completely fill the cyst, and even rupture its wall, and project into the peritoneal cavity. The wall of the original cyst may then be found at the base, or peduncle, of the papillary growth, but may be quite insignificant when compared with the luxuriant development of the latter. Dr. Wilson Fox attributes to the union and fusion of such papillary outgrowths the formation of secondary cysts.

(*c.*) In the above-mentioned variations one cyst preponderates over the others, or seems to have been formed primarily; but in other cases there is a more uniform development of several, so that it appears a section made of many cavities nearly equal in size, the partitions of which may be more or less fused, and showing in their walls the development of new cysts. This appearance forms a transition to a still more uniform arrangement, when the whole of the ovary is found transformed into a multilocular structure, with very numerous closed spaces, mostly minute, not larger than a hemp-seed, containing fluid or gelatinous matter. This change usually affects both ovaries, does not go to an excessive size, and was formerly called alveolar degeneration of the ovary.

The above-mentioned appearances are by no means exclusive of one another. They may occur simultaneously, and by their combination produce an infinite variety and complexity of appearance in ovarian cysts—which is heightened by such variations as are obviously possible—in size, thickness of the walls, and so forth.

Contents of the Cysts.—In some cases this is clear, serous fluid, colourless or yellow; from this all transitions are found, up to thick gelatinous matter, and even more solid colloid material. The fluids always contain albumen and modifications of it, called paralbumen and metalbumen ; also a fibrinogenous substance, like many serous effusions. The above characters also belong to the fluid of simple cysts. The thick, viscid, and gelatinous matters often found in compound cysts are of different chemical constitution, containing mucin ; but whether this is the real constituent of the former colloid masses is still uncertain. These matters are coloured, yellow, brown, or green, in various shades, probably by an admixture of blood. Cholesterine is sometimes found, and also much detached epithelium.

Size of Ovarian Cysts.—As is well known, these form some of the largest tumours ever known in the human body. Specimens weighing 40 or 50 pounds have often been met with. Klob mentions one of 150 and another of 196 (German) pounds in weight, which is perhaps the largest tumour ever described, except the fatty tumour before mentioned (p. 139).

Structure of the Walls.—This point is very important, as it is thought to contain the key to the formation of ovarian cysts. The walls are composed essentially of connective tissue similar to that of the stroma of the ovary, in which are contained very frequently minute cysts, either visible to the naked eye or under the microscope. With these are seen indications of the Graafian follicles or similar structures, which are, as some think, wasting and gradually being destroyed ; according to others, there are many still in a rudimentary stage. Further are structures which many observers regard as the commencement of minute cysts, viz., in the words of Förster, collections of roundish or oval cells, closely pressed together, in the midst of the connective tissue. Some of these are in a state of colloid degeneration, and thus form the contents of a microscopic cyst. The outer cells show transition to epithelium and a fibrous sac.

Masses of developing connective tissue, or sarcoma-like structure, are often seen.*

Another structure to which much importance has been attached is that of glandular tissue, *i.e.*, epithelial cells in a tubular form. This tissue is very abundant in certain ovarian tumours, which have been called *adenomatous*, or glandular. No similar tissue is seen in the mature, healthy ovary, but in the course of the development glandular tubes, described by Pflüger, appear to be steps in the formation of the Graafian follicles. This glandular tissue is seen to pass into a state of colloid degeneration, or to produce colloid substance ; so that it is believed the colloid masses in compound cysts may be formed in this way, and that all transitions may be found up to the largest cavities.

Theory of the Formation of Compound Cysts.—From what has been said, it will be seen that all observers regard the minute cysts formed in the wall of the larger ones as giving the explanation of cyst formation. No recent investigator (except Klob) regards these as Graafian follicles; that is, supposes that compound are formed in the same way as simple cysts. Dr. Wilson Fox thinks that the primary cyst is an enlarged Graafian follicle, but that the secondary cysts are formed in its walls by different processes, chiefly by a kind of papillary growth; also by the transformation of rudimentary glandular follicles, and by free cyst production in the connective tissue. Förster regards it as a process of cyst formation in connective tissue. Klebs (with whom Waldeyer and other recent German investigators agree) derives the secondary cysts from the residue of fœtal glandular structure, and therefore places the formative process in the latter part of fœtal life, and connects them with the glandular tumours before referred to; so that he gives a compound ovarian cystic tumour the name of *adenoma cylindro-cellulare cysticum.* Dr. Ritchie contends for the participation of the ovum. These theories we cannot discuss.

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* Figured by Mr. Arnott in Dr. Barnes' "Diseases of Women," p. 321.

CHANGES OF COMPOUND CYSTS.

Suppuration not unfrequently occurs, and sometimes causes perforation of the wall, followed by peritonitis; or it may open on the outer surface of the body.

Twisting of the pedicle sometimes occurs, and may produce little effect but often leads to inflammation or, by obstructing the vessels, to atrophy.

Wasting, accompanied by thickening of the contents, or even complete solidification, may occur spontaneously. The wall may become calcified.

Combinations of compound cystoid tumours with cancer, or with sarcoma, or with dermoid cysts, sometimes occur.

Dermoid, or Cutaneous Cysts; Proliferative Cysts.—The general characters of these cysts have been already described (p. 157). They are far more common in the ovaries than elsewhere, two-thirds to four-fifths of all the known cases having occurred in these organs. They are generally, if not always simple, and for the most part solitary. The inner wall seems always, to be formed by a structure precisely resembling epidermis, having flattened cells in the more superficial, and round cells in the deeper layers. This stratum rests upon a connective tissue structure, resembling the cutis, which is often elevated into papillæ irregularly arranged; outside all is often an investment of fatty tissue, like the panniculus adiposus. Inserted in the skin are hairs completely formed, sebaceous glands attached to their bulbs, and further sudoriparous glands. Teeth are often contained in great numbers—more than 100 have been counted—and either grow in the unaltered wall or else are imbedded in bony sockets. Bone is also sometimes found, both in the fibrous wall and detached, never having any real resemblance to any actual bone of the skeleton. The remaining contents of the cysts are tallowy or crystalline fat, with cholesterine, detached epidermic scales, and hairs. The fatty matter is doubtless secreted by the sebaceous glands. It will be observed that all these products except bone belong to the epidermis, but in a very few rare cases other tissues, as striped muscle and nerve tissue, have been observed; as, indeed, has also been the case in some dermoid cysts from other parts. These cysts are liable to inflammation and rupture, sometimes discharging externally, or else into the vagina or rectum.

Origin of Dermoid Cysts.—The explanation of these growths is confessedly one of the obscurest chapters in pathology. When occurring in the ovary, they were at one time regarded as the remains of an imperfectly-formed fœtus—a view plainly untenable, since they have occurred before menstruation; or, again, as the inclusion of a second fœtus within the body of the first, which is an unsupported hypothesis. Generally later pathologists have concluded that they must be in some way a result of the normal productivity of the ovaries, exercised without impregnation, in an altogether abnormal way, the process being compared sometimes to the generation by which lower forms of animal life increase, or more specifically to *parthenogenesis*. If these tumours never occurred in other parts of the body, this theory would at least indicate the direction in which an explanation should be sought, but no one would attribute a genetic function to the skin or the mediastinum. It appears to us that a far more hopeful suggestion is that originally made by Dr. Wilson Fox, to the effect that these cysts will be found to arise by the same laws as the ordinary compound cystoid tumours. This comparison, at that time unsupported by observations, has been confirmed by the few cases of simultaneous occurrence of dermoid and compound cysts, especially by a remarkable case described by Flesch, and others adduced by that writer.*

Cancer of the ovary may be either primary or secondary. Primary cancer usually occurs in a form not distinctly scirrhous or medullary, but either, like many cancers of internal organs, of an intermediate hardness, or else showing a different consistency in different parts. It is very often indeed combined with cysts, either simple or compound. We have seen one case of combination of cancer and a dermoid cyst. In the softer forms it grows rapidly, and may form a tumour as large as a child's or a man's head. It comparatively rarely ulcerates, and does not show a very marked tendency to attack the neighbouring organs, though it may form inflammatory adhesions. Not unfrequently it occurs in both ovaries at once. Hard, scirrhous-like forms are very rare. In such specimens as we have examined, we have found a distinct alveolar structure, with cells of epithelial aspect, such as has been described in the first part of this work; the cells are usually rather small than large. In some cases it has been thought that some indication of glandular structure existed, which connects these growths with such as arise from the fœtal glandular structure of the ovary, and with the rudimentary Graafian follicles.

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* Wilson Fox, "Medico-Chirurg. Transactions," vol. xlvii. 1864. Flesch, "Würzburger Verhandlungen," vol. iii. Heft 2, 1872. See also "Trans. Path. Soc.," vol. xviii. p. 190.

Secondary cancer may occur by extension of the disease from neighbouring parts, as the uterus or Fallopian tubes; more rarely from distant organs.

Sarcoma of the spindle-celled variety has been several times observed in the ovary. It sometimes appears to be combined with carcinoma, and in that case it is assumed that the sarcomatous element comes from the stroma of the ovary, the carcinomatous from the epithelium of the Graafian follicles, or the rudimentary forms of those structures. A melanotic tumour, probably sarcoma, has been described.

Tumours of the Ovary.—Fibrous tumours are not very uncommon, and sometimes reach a large size. Rokitansky describes a form of fibrous or fibroid tumour, which originates in degeneration of a menstrual corpus luteum.

Fibroid, or fibro-muscular tumours, like those of the uterus, also occur; but caution is necessary not to mistake for these uterine tumours which have become detached.

Cartilaginous and bony tumours have been described, but were probably only a part of dermoid cysts. Angioma has been observed in one case ("Trans. Path. Soc.," xx. 203). No other forms of simple tumour are known to occur in the ovaries; a remarkable fact, if we consider the presumed reproductive energy of these organs.

ABNORMAL CONDITIONS OF THE FALLOPIAN TUBES.

Inflammation, Salpingitis.—The Fallopian tubes are liable to various forms of inflammatory action. Catarrh and exudative inflammation not unfrequently cause a temporary or permanent closure of their channel, which prevents conception, and may lead to dropsical accumulations and other morbid conditions. Thus the fimbriated extremities may become agglutinated to the ovaries, the broad ligament, or the uterus itself; or obliteration may occur at one or more points within the passage; unless the mucous membrane of the part still patent be deprived of its functions, the continued secretion will cause distension, simulating a cyst formation. We have seen a case of dropsy of the Fallopian tube, in which the distension amounted to about five inches in diameter. At other times, according to Rokitansky, several saccular dilatations form between the separate angles and projecting duplicatures of the tubal parietes, and give rise to an imperfectly loculated pouch, which, as in the former case, may contain mucous matter of a more or less purulent character, or fluid of an heterogeneous constitution. These morbid contents are sometimes poured into the uterus, probably in consequence of the occlusion only having been effected by inspissated mucus; a less favourable issue is rupture of the sac, and effusion of its contents into the abdominal cavity. Morbid fluids can also enter the peritoneum by the apertures of the fimbriated extremity. This occurs in acute inflammation, both puerperal and non-puerperal, producing peritonitis. It should be remembered that uterine injections, or air, may by this means find their way into the peritoneum.

In some cases of dilatation the uterine orifice of the tube is distinctly unclosed, and obstruction appears to be caused, as in intestinal obstructions, by flexion.

Hæmorrhage into the tubes is occasionally seen, perhaps coming from the ovary. It has usually undergone degenerative changes when observed. This may be one of the causes of dilatation.

Tubercle of the Fallopian tubes is much more common than that of the ovary, and is sometimes a primary disease. We find the tubes filled with white or yellowish pasty matter, proceeding from degenerative catarrhal inflammation of the mucous surface. The walls are thickened and indurated, the canal sometimes irregularly dilated and tortuous. Tubercles may be seen, it is said, on the mucous surface. In such cases as have come under our notice, we have found them in the walls or on the peritoneal surface, where they may be the starting point of tubercular peritonitis. This condition may be combined with a similar disease of the uterus, or occur independently. The lymphatic glands generally become affected. Small cysts frequently form on the fimbriated extremities of the tubes. They have thin walls, and are not usually so large as a pea.

A congenital structure, the so-called Hydatid of Morgagni must not be confounded with this. It is a cyst as large as a pea or a bean, attached by a pedicle, one or two inches long, or more, to the fimbriated extremity of the tube. It is said to occur in about one body in four, and is explained as the remnant of Müller's duct, out of which the Fallopian tube is formed.

ABNORMAL CONDITIONS OF THE BROAD LIGAMENTS.

The only anomaly which requires notice here is the occasional development of cysts from the body called the parovarium, or organ of Rosenmüller, a mass of tubular structure which lies near the ovary, between the layers of broad ligament, and is, in development, a relic of the fœtal kidney. Minute cysts are not very uncommon in this situation; but occasionally some are formed, which in their size rival ovarian cysts, and may, without care, be confounded with them. Some fibrous tumours of the broad ligament, which have been attributed to the ovary, are believed to originate in this body. Cysts may occur on the broad ligament, unconnected with the parovarium.

ABNORMAL CONDITIONS OF THE MAMMÆ.

The physiological condition of the breast is subject to so many and important changes, that it is needful to bear these in mind in all cases of disease of the organ, taking into consideration at the same time the close connection which exists between the state of the pelvic procreative organs and the mammæ.

Thus, the anatomical conformation of the breast varies materially at different times of life. In the infant, for instance, and until puberty, almost the only structures to be distinguished are the minute ducts converging to form the short, straight tubes of the nipple, and these are connected and supported by a variable amount of young connective-, and wavy white fibrous and elastic tissue. Very little fat is present, very little vascularity, and even the essential glandular structure, the minute cæcal duct-terminations, whose epithelium furnishes the milk, are almost wholly wanting in youth. The tubes themselves are often mere cylindrical collections of epithelium with clubbed extremities.

After puberty a considerable enlargement of the breast attends the awakened functional activity of the ovaries, and the vascular excitement now present may be periodically augmented at the catamenial periods. It is not, however, until conception has taken place that the glandular structure becomes fully developed. The cæcal terminations of the ducts now dilate into acini, lined with epithelium, which early in pregnancy shows that disposition to engorgement with oil particles and rapid proliferation, which subsequently produces, first the cells of colostrum, and afterwards the perfect milk. At the same time the division of the organ into separate lobes becomes more clearly marked, and this lobulation remains henceforth a striking peculiarity of the breast, until the cessation of the catamenia at the climacteric period. At this time retrograde changes set in, the glandular structures become inconspicuous, and much fat is found separating the atrophied lobes of the breast.

Now, at all the periods of life the mammæ are subject to distinct changes, which may be directly traced to slight failures or excesses of normal physiological processes.

In newly-born infants a slight attempt at secretion of milk, attended with fulness, redness, and tenderness, and some escape of serous fluid from the nipple, is extremely common, and may be readily stimulated to inflammatory changes and even to suppuration by the injudicious efforts of nurses to promote the flow of milk by manipulation and friction.

At puberty, too, the new excitement and rapid development may often be attended with much uneasiness, tenderness, and neuralgic pains, and care should be taken to avoid anything like injurious pressure upon the enlarging organs. In boys, the slighter changes at this period not unfrequently take the form of a tender induration surrounding the nipple, attended with some heat and pain, but lasting for so short a time as rarely to be brought under the notice of the surgeon.

In the period following puberty, and especially during lactation, irregularities in the exalted functional activity are prone to give rise to general hypertrophy, or into local hypertrophies which assume the form of adenoceles, and more especially into the inflammations which are so frequently forerunners of abscess, either acute or chronic, and into cystic tumours.

It is in the period of involution, or the commencement of the atrophic changes, which mark the cessation of active physiological processes, that the graver forms of breast disease—and notably scirrhus—are met with.

Inflammation of the breast, although it may be met with at all ages as the result of injury, or as one of the manifestations of those manifold conditions which are grouped together under the head of struma—in which case the inflammatory action is usually very slow and suppuration long delayed—is generally a complication of lactation. Any interference with the due performance of this function is prone to lead to inflammation and abscess. Hence one very common exciting cause is the sudden cessation of suckling, or allowing the breasts to become engorged with milk. Prolonged lactation, also, with its drain upon the general health, is apt to set up inflammation. A very common accompanying condition, upon which Mr. Birkett lays much stress, is some defect in the nipple, causing either soreness or blocking of the ducts. It produces congestion in one or more parts, accompanied by swelling, interstitial effusion, condensation, and finally, if the disease be not arrested, suppuration and abscesses. The glandular texture itself, or more frequently the intercellular tissue, is the primary seat of lesion; the lacteal secretion frequently continuing during the inflammatory process, and even after a chronic state of induration and enlargement has been established, which has led to the removal of the organ. When the inflammation is confined to the true gland structure, the resulting tumefaction is irregular and lobulated, and deeper seated than when the interstitial or cutaneous tissues are mainly involved. When suppuration is established, it may be limited to one spot by adhesive inflammation, and the abscess be evacuated by pointing, as it usually does, near the nipple; or, a burrowing sinuses form, which may extend to a considerable distance. The symptoms of mammary abscess do not differ materially from those of suppuration in other parts. Besides the constitutional disturbance, which may be very severe, one or more lobes of the breast become indurated, swollen, and very tender and painful; the skin after a time betrays the mischief beneath by its redness and œdema, and finally the abscess points at one or more places, and slowly drains away.

The practical surgeon distinguishes three kinds of abscess about the breast, viz.: the true mammary abscess, in which the glandular structures are involved, the superficial abscess betwen the skin and the gland, and the post-mammary abscess, in which the suppuration has its seat in the cellular tissue behind the breast, pushing the organ forward in a remarkable manner, and after a long time discharging either through the breast or, oftener, by its side. The chronic induration, either following an abscess or with little tendency to suppurate, is often very difficult to distinguish from a new growth in the breast.

Hypertrophy.—True hypertrophy sometimes attains a very considerable size in unmarried females, and appears to be an indication of a generally precocious tendency. Commonly both breasts are affected; but occasionally one is inordinately developed. A temporary enlargement of the gland very commonly accompanies menstruation; it may occur periodically, even long after the cessation of this function, as in an old lady of eighty-five, who was under our care, and who was subject to this phenomenon regularly every month. A permanently hypertrophic state is induced by lactation, the period which directly or indirectly gives rise to many of those morbid conditions to which the gland is liable. During lactation, large accumulations of milk frequently distend the entire system of ducts, or a single portion; in the latter case, a fluctuating tumour may result, which will scarcely disappear without surgical interference. It is stated, that as much as ten pints of milk have been evacuated from a swelling of this description. It appears to be generally owing to an atonic state of the mammary ducts, similar to the condition of the efferent channels in the nipple, causing a non-retention of the secretion. The lactiferous tubes are occasionally found to contain sebaceous-looking matter, phosphatic concretions, and other products, which have been attributed to the effects of chronic inflammation (College of Surgeons, Nos. 2,743; 2,744; 2,747; 2,748); but unless there is a coincident change in the coats of the ducts, it is probable that these matters are the residue of an effusion of milk which has been long retained, and in which a partial absorption has taken place.

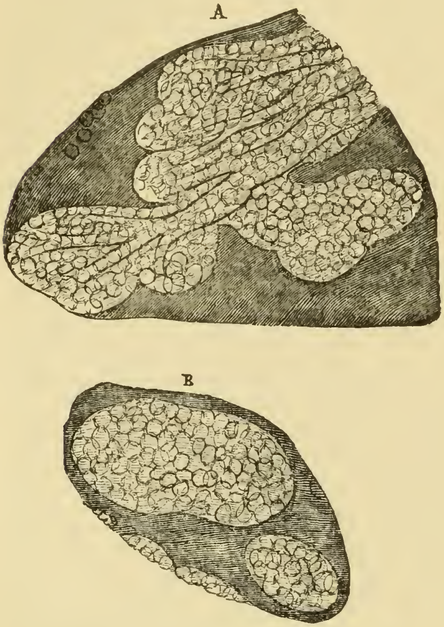
TUMOURS OF THE BREAST.

Notwithstanding the attention which has been specially given to tumours of the breast, their classification is still involved in much confusion. We believe the following to be the most clearly defined species of tumours:—(1) Lobular hypertrophy; (2) Adenoma, or cylinder-celled epithelioma; (3) Cysto-sarcoma; (4) Cancer, including medullary, scirrhus, and colloid; (5) Sarcoma; and (6) Simple tumours.

Lobular Hypertrophy.—By this is meant partial hypertrophy of the glandular structure, or the partial formation of a tumour. Although tumours of this description ordinarily remain in connection with the proper gland tissue, they sometimes appear to be altogether isolated.

In a small tumour of this description, removed from the breast by operation on the supposition of its scirrhous nature, and of its being unconnected with the gland, which we had an opportunity of examining, the microscope revealed well-marked ducts and lobules, in no essential feature differing from ordinary mammary tissue. We have since repeatedly examined the structure of mammary tumours, which were regarded as malignant, and found them to consist of follicular structure, filled with epithelial growth. Mr. Birkett describes this form of tumour as presenting to the naked eye a granular appearance of a white, rosy, or red colour, dependent in a measure upon the time it has been exposed to the air; it is lobulated, divisible into the most minute lobules, attached by a prolongation to the breast, and invested by a fibro-cellular envelope continuous with the proper fascia of the gland. The lobules are connected by common areolar tissue, and the growths vary in size from that of a marble to that of a child's head. This might also be called adenoma, being a true glandular tumour.

Fig. 183.



Lobular hypertrophy of mamma.

A. Section showing the entrance of duct.
B. Cross section, resembling cystic disease.

Cylinder-celled epithelioma, or true Adenoma, is described by Billroth and Rindfleisch. It is produced by proliferation of the glandular epithelium, and forms an alveolar structure, in which cylindrical epithelium is contained, like that of the normal glandular acini, but arranged in a disorderly manner, so as to fill up the cavity, instead of remaining ranged along the walls. It ends in fatty degeneration and formation of atheromatous cysts, but the cystic dilatation of the ducts is not notable. It is plain that this tumour constitutes a transition from simple glandular hypertrophy to cancer, resembling the former in the character or shape of its cells, the latter in their arrangement. It is a rare form of tumour; we have had no opportunity of examining it.

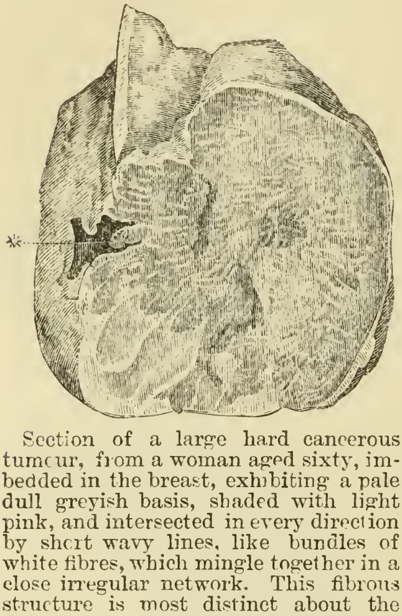
Cysto-sarcoma Mammæ; Sarcoma Adenoides.—This form of tumour is not so uncommon as that just described, and has been the subject of numerous histological investigations. Nevertheless, its anatomy and history are by no means clearly made out; they have sometimes been confounded with simple hypertrophy. The new growth forms a nodular lobulated mass, of elastic or hard consistency, involving a larger or smaller part of the mammary gland; usually solitary, but sometimes multiple, and affecting commonly only one breast. It grows very slowly, and rarely ulcerates. The structure of these tumours is complicated, consisting partly of solid sarcomatous tissue, partly of cysts, and partly of glandular tissue. The solid tissue is plainly an outgrowth of the fibrous tissue, which forms an important constituent of the mamma, especially in early life. It consists of sarcomatous tissue; that is of round or spindle cells, with a considerable amount of intercellular substance and fibre. According to Billroth this is the essential part of the tumour. Portions of glandular structure, both acini and excretory ducts are also seen, and it has been generally thought that part, at least, of this is newly formed gland-tissue; so that the tumour must be called a glandular tumour, or adenoma (p. 152). Billroth, however, contends that the gland structure is nothing but the remains of the normal gland; though he admits that the hypertrophy of the matrix must entail some enlargement of the acini. The usual appearance is to find slits, or very narrow ramified cavities, lined with cylindrical epithelium—an appearance which may be explained by compression of the acini, but also, as we shall see, otherwise. The cysts vary in size, some being microscopic, and appear to be formed by dilatation of the acini, from an obstruction of their outlets. They are lined with cylindrical epithelium, and contain serous or mucous fluid. Very often these cysts are complicated by proliferating growth into their interior, which may almost fill the cavity, and thus give rise to some of the narrow ramified cavities, lined with epithelium, above described. The minute cysts in the stroma, no doubt, go on multiplying and increasing in size till a very complicated structure results. In fine, we must think that there is in these tumours a new production, both of glandular tissue and of stroma, though one may often preponderate over the other.

CANCER OF THE MAMMA.

Cancer affects the breast more frequently than any other organ of the body. All the varieties of carcinoma have been met with here; scirrhus is, however, by far the most frequent form in which it occurs primarily. The encephaloid variety may be primary, but is more commonly engrafted upon the former; the colloid form is the most rare. When associated with other cancers, that of the mamma is stated by Dr. Walshe to be invariably primary, except in those rare instances when the disease spreads from the lymphatic glands or superjacent skin.

Scirrhus occurs in the form of a hard, lobulated tumour, imbedded in the adipose tissue of the gland, causing adhesion to the skin, and retraction of the nipple; it is at first somewhat movable, but soon becomes firmly adherent to the subjacent parts, involving more or less of the gland tissue, the thoracic muscles, and the adjoining lymphatic glands. Instead, however, of occurring as an isolated tumour in the first instance, it may from the commencement appear as an infiltration of the various structures of the part; it will in that case be ill-defined, sending out branches into the adjacent tissues, and involving in its mass the lacteal tubes and lymphatics. These become contracted and flattened into bands, which give a peculiar appearance to this form of mammary cancer. Scirrhus is not at first accompanied by pain, hence its existence is often accidentally discovered when it has already reached the size of a marble or a pigeon's egg. Even when quite small it is usually very hard. Ulceration of the skin in the vicinity of the nipple supervenes; the edges of the sore are raised, everted, and puckered; a purulent, ichorous fluid is secreted, from a bluish-red, eroded surface, offering a faint and fœtid odour; bleeding often ensues, and the patient sinks from exhaustion.

Fig. 184.



Section of a large hard cancerous tumour, from a woman aged sixty, imbedded in the breast, exhibiting a pale dull greyish basis, shaded with light pink, and intersected in every direction by short wavy lines, like bundles of white fibres, which mingle together in a close irregular network. This fibrous structure is most distinct about the centre of the mass; its exterior appears more homogeneous.

* The retracted nipple.

(St. Barthol. Museum, xxxiv. 14.)

Minute Characters.—The minute structure of scirrhus has been already described (p. 175), the characters of this form of cancer being chiefly derived from the breast. The cells, when undegenerated, are large, flat, and angular, and even in very hard specimens often numerous; but are also very liable to atrophy, so that we may find only wasted alveoli with the remains of cells. The growth always appears to begin in the gland, but spreads beyond it by continuous infection of the surrounding tissue. We then find cancerous alveoli scattered in the neighbouring parts. Waldeyer regards these alveoli as sections of cylindrical processes spreading outwards from the gland, and supposes all the cellular part of the cancer to be an outgrowth from the glandular epithelium of the mamma; others suppose that the cancerous cells are formed out of the connective tissue passing through a stage of small-celled infiltration, as seen in Fig. 43 (p. 186). Waldeyer urges, on the other hand, that this small-celled infiltration is quite unconnected with the growth of the epithelial or special cells of the cancer, except in so far as the latter act as an irritant. The question is still *sub judice*, and we can express no opinion about it, except that our experience agrees with Waldeyer in being unable to trace a transition between small-celled infiltration and formation of alveoli. The fibrous stroma appears to be the result of the former process. It varies much in amount. The axillary lymphatic glands are commonly swollen, hard, and infiltrated with the same carcinomatous products as the mamma; their affection appears to be coincident with the implication of the skin at the primary seat of injury. The pectoral muscles, the ribs, and costal cartilages are also found secondarily involved to a greater or less extent; a secondary affection of the pleura and the lung is not unfrequent. Towards the termination of the disease, from direct interference with the venous circulation, œdema of the extremity of the affected side is liable to supervene.

Billroth and some of the German pathologists distinguish the very hard and early atrophic forms of hard cancer as true scirrhus, and call others in which the atrophic tendency is less marked, simple cancer or *carcinoma simplex*. This distinction is not usually adopted in this country, and the question is one mainly for practical surgeons; we only mention it here to avoid the confusion of synonyms.

Medullary, or Soft Cancer.—This is said to occur earlier in life, and to run a more rapid course than scirrhus, out of which it is sometimes developed, and to give rise to tumours of great size. It is soft, easily breaks down, and is particularly liable to ulceration, sloughing, or vascular growth, in the form of what was at one time called fungus hæmatodes. The structure is more or less vascular, often white or cream-coloured, and brain-like or encephaloid in appearance. The minute structure of these tumours is chiefly cellular, the fibrous stroma being unimportant. The development is said to show unmistakeable continuity with the glandular structure of the breast, commencing with solid outgrowths of glandular epithelium.

Colloid Cancer of the Breast.—This form of tumour is extremely rare. In most of the cases which have been observed it has been combined with another form of cancer, either medullary or scirrhus,* and even in specimens where no other kind of tumour is obvious, minute examination may show that the colloid mass results from the degeneration of groups of cells contained in cancerous alveoli (p. 176). It produces very marked local infiltration, and may be reproduced in distant parts. We have seen very extensive infiltration and ulceration over nearly the whole of the chest. A similar case is described by Doutrelepont.†

For the clinical history of mammary cancer, the influence of age and other predisposing causes, and for statistics, we must refer to works on surgery.

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* Croft, "Trans. Path. Soc.," vol. xxiii. p. 265, pl. x.

† Langenbeck's "Archiv. für Klin. Chirurgie," vol. xii. p. 551, pl. ix.

Sarcoma.—A true spindle-celled sarcoma has been seen in the mamma, but is rarely uncomplicated, there being generally either cystic production, or sometimes an intermingling of cancer. It often recurs after removal, and may come in the end greatly to resemble the soft cancer in external appearance,* and to be reproduced in distant parts. No doubt such tumours were formerly called cancer; indeed, the name carcinoma fasciculatum of Joh. Müller, which means spindle-cell sarcoma, was taken from tumours of the breast.

Myxoma is sometimes observed, but was probably formerly confounded with colloid cancer. It may recur and even be reproduced in distant organs.†

Fibroma.—Simple fibrous tumours are not very common. They arise, according to Virchow, from a hyperplastic process in the fibrous stroma of the mamma, in which the glandular structure disappears. This may be general or partial, the former causing general induration of the glands. A peculiar form of tumour is Virchow's *fibroma papillare intracanaliculare*, formed by papillary growths within the lactiferous ducts. We have seen one case, and another has been lately described.‡ True fibromata seem sometimes to contain cysts.§

Enchondroma occurs in the breast seldom, if ever, unmixed, but usually combined with myxomatous and sarcomatous tissue; more rarely with cancer. But it is in any form very uncommon. Sir A. Cooper described a case which was partly bony.

Lipoma.—Isolated fatty tumours are not unfrequently formed in the adipose tissue surrounding the gland. General hypertrophy of the same tissue produces the appearance of enlargement of the gland. The production of fat may indeed be combined with a general interstitial fibrous hypertrophy, producing apparent enlargement, though wasting of the gland substance; but it may also occur in cases of actual wasting of the breast, from scirrhus or fibroid induration.

Echinococcus cysts have occurred in the breast, but are excessively rare. One is reported in the "Pathological Transactions" by Mr. Bryant (vol. xvii. p. 276).

Tubercle has not been observed to occur in the breast.

Syphilitic growths in the form of gummata are spoken of by some authors, but the descriptions for the most part require confirmation.

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* Anderson, "Trans. Path. Soc.," vol. xxiii. p. 254, pl. viii.

† See a remarkable case in "Trans. Path. Soc.," vol. xxiii. p. 275, and vol. xxiv. p. 120, pl. iii.

‡ Virchow, "Krankh. Geschwülste," vol. i. p. 342. De Morgan, "Trans. Path. Soc.," vol. xxi. p. 353. (?)

§ "Trans. Path. Soc.," vol. xxiii. pp. 258, 260.

THE MALE MAMMA.

The male breast is occasionally the seat of non-malignant and malignant growths. We have ourselves met with an instance of the former in a gentleman aged twenty-one, which to the touch closely resembled one of hypertrophy of the mammary gland; it was of the size of a shilling, felt semi-cartilaginous, as if composed of lacteal ducts, and was adherent to the skin of the nipple. The individual had perceived it six weeks before applying for advice; it gave no pain, and four months later we were informed that it had almost disappeared without any active treatment being pursued. This is in accordance with the structure of the male mamma, which Sir A. Cooper has shown to resemble the female gland, though in a rudimentary state. Nor could we otherwise account for the well-authenticated cases of the secretion of milk by men. Mr. Birkett gives delineations, showing the male gland to have all the essential elements requisite for the performance of the function. Mr. Stanley* relates the case of a man, aged forty-five, who was affected with cancer of the right humerus, secondary to cancer of the right breast. Cruveilhier states that three cases have come under his observation, one of which is delineated in his atlas.† In the College of Surgeons (prep. 2,791) there is the section of the breast of a man with a very vascular ulcer, five inches in diameter, probably originating in a lens-shaped, hard cancerous tumour, or a degeneration of the skin and mammary gland. The monographs on diseases of the mamma also contain records of simple cysts, compound cysts, and encysted tumours, occurring in the male breast; but they belong to the mere curiosities of medical experience.

Pleiomazia, or excess in the number of mammæ, has been observed very rarely in men; more commonly in women.‡

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* "A Treatise on the Diseases of the Bones," by Edward Stanley, F.R.S., 1849, p. 255.

† "Anatomie Pathol.," Livr. xxiv.

‡ Murchison, "Trans. Path. Soc.," vol. xvii. p. 426.

CHAPTER XLIII.

MORBID CONDITIONS FOLLOWING AND PRECEDING PARTURITION.

It is immediately after parturition that the uterus, which during pregnancy has become, as it were, the focus of the entire system, and having completed the great cycle of its duties, is required to lapse into its previous dormant state,—it is while the organ is yet the seat of increased vascular action, and its proper functions may be said to have ceased, that morbid influences are received with facility, and produce destructive and often rapidly fatal consequences. After the enormous evolution which the uterus during pregnancy undergoes, it has, when parturition has occurred, to go through changes in the way of involution which are sometimes imperfectly effected. There are two states which are more particularly liable to supervene immediately after parturition, which are each of them a source of danger, by the hæmorrhage they give rise to. The one is atony or defective contraction; the other, spasm or irregular contraction of the uterus. In the one case we find the uterus maintaining its dilated condition, its walls are flabby and soft; in the other, various irregular forms, to which the term hour-glass contraction has been applied, present themselves.

PUERPERAL INFLAMMATIONS.

The inflammations occurring in the puerperal state may be classified as local and general. The latter constitute what is generally known as puerperal fever, though this term is used with a certain amount of laxity and sometimes applied to conditions properly described as local diseases.

Local Inflammations.—These, when occurring after parturition, have considerable similarity to those which occur in the unimpregnated uterus. They may be described under the corresponding heads of Puerperal Endometritis, Metritis, Perimetritis, and Parametritis; with corresponding inflammations of the ovaries and Fallopian tubes.

Puerperal endometritis chiefly affects the fundus and body of the uterus, the cervix being little affected. Three forms or degrees of it are distinguished by Rokitansky.

In the first the uterus is usually well contracted; the inner surface is covered with purulent or mucous fluid, and where the submucous tissue is laid bare a greenish or yellowish albuminous mass is seen diffused among the muscular fibres. The remainder of the uterine tissue is simply somewhat œdematous.

In the second or diphtheritic form the mucous membrane is more conspicuously swollen and injected, and easily breaks down into a whitish or brownish slough, which may hang on the surface in flakes. In other parts are croupous false membranes, not usually very extensive. There is, as in the other form, albuminous matter in the muscular bundles of the uterine walls. This condition may penetrate to the deeper tissues, and involve the entire thickness of the uterus, which will then, also, be more or less softened and discoloured, infiltrated with a low sanious product, and even converted into a mere pulp. The dirty-coloured, brownish, flocculent matter that is found on the inner surface of the uterus is doubtless in part composed of the remnants of the decidua, to the decomposition of which Klob attributes an important share in the production of puerperal endometritis. With regard to the nature of the false membrane, it should be stated that it somewhat resembles that of diphtheria of the throat; but some pathologists look upon it as connected with a vegetable fungous growth, or associated with low organisms of the bacterial type. Some degree of suppuration and softening of the uterine walls may also be present.

The third degree of the disease described by Rokitansky is the septic or putrescent form, rarely seen. It is said to differ from ordinary gangrene. The internal surface of the organ is covered with a thin, opaque, or more dense product, varying in colour from pale green to dark brown, beneath which the tissue, to a greater or less depth, is converted into a similar pulp. Small abscesses are sometimes found in the muscular tissue, without any perceptible change in the surrounding parts; generally, however, the structure of the muscular fibre is entirely destroyed, and the consistence of the organ so altered that it takes the impression of the surrounding portion if any pressure is applied to it. Certain portions of the organ show complete necrosis; there is at the same time intolerable fœtor of the parts.

Endometritis readily extends along the Fallopian tubes, and the inflammatory process is thus conveyed directly to the peritoneum; hence the frequency of peritonitis following this affection. It is frequently complicated with metritis, and gives rise to thrombosis of the uterine veins, or inflammation of the lymphatics. It may also prove fatal by setting up a general septic condition, even without the production of abscess or inflammation in distant parts.

Metritis, or inflammation of the substance of the uterus, is usually dependent upon the affection just described. The muscular substance is found infiltrated with inflammatory cells, and ultimately breaks down into suppuration with the production of abscesses, which, as in corresponding conditions of the kidney, are found mostly in the outer parts of the organ. In this way a large portion of the uterus may be destroyed. The affection is usually accompanied by thrombosis and puriform softening of the clots. It readily extends to the surrounding tissues, producing local peritonitis and subperitoneal abscesses. They most frequently start from fissures and ruptures of the cervix, which become covered with false membrane.

Inflammation of the Ovaries and Fallopian Tubes.—These parts are very commonly affected in puerperal inflammations. The tubes, doubtless, become inflamed by direct continuity with the surface of the uterus. In *metro-salpingitis*, as this affection may be called, the lining membrane of the tube is found injected with catarrhal inflammation. and showing profuse suppuration, or the cavity may contain sanious or putrid matters. It is needless to point out how easily these conditions are propagated to the peritoneum. The inflammation thus produced is often only partial or limited to the vicinity of the tubes.

Puerperal oophoritis does not notably differ from simple inflammation already described; it is usually suppurative.

SECONDARY AFFECTIONS.

The morbid conditions above described are the primary inflammations of the puerperal state. We have now to consider the secondary affections of the surrounding parts. These affections are not only important in themselves, but as indicating the means by which the local puerperal inflammations produce a general blood poisoning, pyæmia, or puerperal fever. The lymph and blood-vessels must be first considered, then the adjacent serous cavity of the peritoneum.

PUERPERAL LYMPHANGITIS.

The lymphatic vessels very frequently become affected in puerperal inflammations, and always in consequence of endometritis or metritis. The first change perceived in them is, according to Klob, dilatation of the uterine lymphatics. These vessels are seen in a varicose state on the serous surface of the uterus, and containing a lax fibrinous coagulum, which in many of the vessels is softened into a puriform liquid, so that when one of these is opened it looks like an abscess. The coagulation of lymph is an altogether abnormal phenomenon and depends on the uterine inflammation. Inflammation of the lymphatic glands connected with these vessels is also often seen; and in very severe cases even the ductus thoracicus has been found to contain a similar thrombus. Complete coagulation in the vessels would, of course, block the road to the poisonous matter, but the softening thrombus no doubt facilitates the passage of morbid poison into the blood.

Inflammation of the lymphatic vessels appears to be excited by the presence of the thrombus. They show a somewhat reddish tinge, though not from actual injection, and their inner coat proliferates to form young cells or pus. There may, however, as Klob believes, be a lymphangitis without previous thrombosis.

The consequences of these lymphatic inflammations may be a general state of what is roughly called pyæmia, but especially here shown by inflammations of the peritoneum, and less constantly other serous membranes, enlargement of the spleen and liver; but never metastatic abscesses in the lungs.

PUERPERAL THROMBOSIS AND PHLEBITIS.

The formation of clots, in those of the uterine veins which are connected with the placental attachment, seems to be a normal process and necessary to prevent hæmorrhage. From these clots it is always possible that coagulation may spread to the neighbouring veins, viz., the plexus utero-vaginalis, the plexus pampiniformis, and the twigs of the internal iliac vein. In this way the simple pelvic thrombi, which are evidently very common, are produced.

Beside direct continuity with the veins in the placental attachment of the uterus, pressure of the pregnant uterus seems to be a cause of thrombosis. The coagula thus formed are seen mostly in the common iliac or its tributaries, and the external and internal iliac. These coagula are evidently most likely to lead to the coagulation in the veins of the legs, constituting *phlegmasia dolens;* though it appears that this coagulation has in some cases been traced also to continuity with the placental coagula. Another cause of coagulation is the dilatation to which many of the veins and plexuses will have been subject during the pregnancy.

Dr. Davis deserves the credit of having first shown that Phlegmasia alba dolens depends upon thrombosis, or (as it was then called) inflammation of the crural vein; Dr. Lee first succeeded in tracing the uterine origin of this affection anatomically; he demonstrated the "inflammation" commencing in the branches of the hypogastric vein, and subsequently extending from them to the iliac and femoral trunks of the affected side. The cellular tissue surrounding the vein participates in the swelling, and that, as well as the impeded return of the venous blood to the heart, gives rise to much œdema of the limb, and condensation of all the tissues. This may be followed by suppuration, or sloughing; it may terminate in a complete cure, by resolution; or, in a partial recovery, with obliteration of a portion of the vein, and permanent induration of some of the soft parts. In a case examined twenty-one months after the attack, Dr. Lee found the external iliac vein, with its subdivisions, and the upper part of the femoral, converted into a ligamentous cord, so that it could only be distinguished from the surrounding cellular tissue by careful dissection. No trace of the entrance of the common iliac into the cava could be made out. The left side has a greater tendency to become affected than the right. The thrombi formed by either of these processes, when no inflammation is present, are quite innocent. They may gradually be removed or changed, in the manner described in speaking of thrombosis (p. 391), without producing any symptoms. The only danger arising from them is that of embolism; *i.e.*, detachment of a mass of clot which may interfere with the heart, or block up the pulmonary artery, as before described.

It is well known that such accidents do happen in the puerperal state. But in consequence of puerperal inflammation a different series of changes is gone through by the thrombi, and the consequences of any detachment of masses from them will also be different. The thrombus passes into the condition of puriform softening; the wall of the vein frequently becomes inflamed, and thus actual suppuration within the vein may occur. Fragments from a clot thus altered will, if they reach the pulmonary artery, set up abscesses there, and probably also produce general pyæmia. This they do partly because they come from an inflamed part, but also because in those uterine inflammations which are specific, the emboli are the vehicles of the specific poison.

Thrombosis and phlebitis thus supply one channel by which poisonous matters can enter the system. They are in fact, as said before, part of the machinery of pyæmia.

PUERPERAL PERITONITIS.

The serous membrane of the abdomen is very frequently attacked by inflammation in the puerperal state. Peritonitis occurs primarily or secondarily, and is, undoubtedly, the lesion most commonly associated with puerperal fever. It may be limited to the surface of the organ, and more particularly to the part surrounding the neck, or it may involve more or less of the entire sac.

The membrane never exhibits much vascularity, and in the low typhoid forms there is a remarkable absence of congestion and redness. In the more sthenic forms which approach to the character of ordinary peritonitis, the greater vascular action is accompanied by the production of lymph and pus of a healthy appearance, adhering to the surfaces, and matting them together. The ordinary character of the exudation, however, is a copious effusion of an aplastic character; the abdomen then contains from a few ounces to several quarts of serum, of a dirty-yellow, greenish, or brownish hue, in which flocculent particles of lymph are floating, while but small patches of a thin non-coherent exudation are observed on the peritoneal sac.

The smell of the fluid is also distinctive; it will be recognized when once noticed, as it differs from anything met with in the human body, in health or disease. The fluid is described by the older authors as of a creamy character, hence the long-prevailing fallacy that it was connected with an actual metastasis of milk, which was in a measure supported by the failing supply of the mammary secretion observed as one of the first symptoms of the disease. The serum contains a comparatively small portion of albumen, offers an acid reaction, and is said to possess a very salt taste.

Causes of Puerperal Peritonitis.—This is in most cases the consequence of the entrance of inflaming matter from the adjacent organs. This may come directly through the Fallopian tubes; or indirectly by the lymphatics. Dr. Barnes suggests that there may be an actual transudation of liquid through the walls of the uterus, in consequence of the great tension to which they are subject in parturition. In some cases direct propagation from the serous covering of the uterus must be assumed. It is doubtful whether peritonitis occurs, in the puerperal state, with a perfectly normal state of the uterine organs. It may occur as a simple peritonitis, without any further symptoms of puerperal fever; or may be one of the manifestations of this disease.

PUERPERAL FEVER, OR PYÆMIA.

The affections described above need not all be regarded as parts of any general disease. The uterine organs, like others which have been exposed to strain and injury, are very likely to become inflamed. In the same way inflammation may be set up in the peritoneum, as it is by hernia or obstructed bowel.

But in the special diseases connected with the puerperal state a certain order and connection of the morbid conditions is seen, which makes the process strictly analogous to pyæmia. Some inflammation or specific morbid process, affecting the uterine organs, is the primary or local disease which, distributed by means of the veins or lymphatics, produces secondary or general infection. The order appears to be as follows.

Endometritis is usually the primary process, and is often combined with metritis. On this follow most frequently lymphatic affections, lymphatic thrombosis, lymphangitis, or lymphadenitis. These are the sequelæ most frequently seen in puerperal epidemics, and may be compared to surgical erysipelas, or diffuse cellulitis.

The blood is then infected by means of the diseased lymphatics; no mechanical transference of emboli takes place, but an injection by means of fluids. The secondary affections are inflammations of serous membranes; parenchymatous changes in the liver and kidneys and other organs, but no pulmonary abscesses.

Much less frequently, according to Klob, is the uterine inflammation followed by thrombosis and inflammation of the veins. These affections will, however, as in other forms of pyæmia, lead to secondary abscesses in the lungs and other lesions. They are, according to Klob, rarely combined with peritonitis. The lymphatic and venous affections may, of course, be combined.

Specific Theory.—While some investigators hold that the morbid products introduced into the circulation, by the means just described, owe their poisonous character simply to their coming from parts either inflamed or septic, others hold that there is a specific poison which is conveyed in the one case by the lymphatics, in the other by the veins. This poison has been also asserted to be always attached to minute organisms; viz., micrococci or bacteria. This theory is founded on very careful observations by Rindfleisch, Klebs, Heiberg, and others. Having been able to confirm some of Heiberg's observations in pyæmia, we are led to attach much value to his observations in puerperal fever, a malady which we have had little opportunity of studying.*

The minute micrococci, or spherical bacteria, which have been found by Klebs on the surface of infected wounds, are seen also in the diphtheritic false membrane spoken of as occurring in puerperal endometritis. They have been further traced by Klebs and Heiberg into the lymphatics—both the larger trunks and the smaller vessels distributed on serous surfaces—and in lymphatic glands; also in the softened puriform thrombi of veins, and in the heart. Further, they have been traced in the secondary affections, viz., in the pulmonary abscesses, and in the abscesses of other parts. In serous inflammations they were found in large number in the exudation, sometimes making up even a larger mass than the pus and fibrine, and also in the lymphatics of the membrane. It appears then they can be traced through all the channels by which the morbid poison passes, from the seats of primary inflammation into the system generally, and into the secondary morbid processes; wherever the morbid poison can be traced there are the bacteria also. There remain, of course, the further questions where do these organisms originate, and how do they reach the wounded surface of the uterus; but the bearings of these observations on the contagious character of puerperal fever, and on its distribution in the body, are obvious. For a discussion of the questions connected with the contagious or epidemic character of the disease we must refer to works on obstetrics.

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* "Die Puerperalen und Pyämischen Processe," von Hjalmar Heiberg. Leipzig, 1873.

DISEASES OF PREGNANCY.

The consideration of the morbid processes complicating parturition is appropriately followed by an account of the diseased conditions met with in the placenta and in the ovum ; we shall, at the same time, touch upon extra-uterine pregnancy.

THE PLACENTA.

The placenta varies much in size within the limits of health. Its position differs also considerably without inducing any detriment to the mother or child ; but when placed near or over the os uteri, the frequent hæmorrhages that occur endanger the life of both. The umbilical cord, instead of being attached to the centre of the placenta, is sometimes inserted at the edge ; this gives rise to what has been termed the battledore placenta, a deviation which, though not in itself perilous, may become so by rough manipulation after the birth of the child. The same applies to those cases in which the vessels of the cord are divided before they reach the placenta or are inserted into the membranes. Other irregularities of the cord consist in its being excessively short or extravagantly long, and in its being tied into knots. The extremes of length on record are two inches and fifty-seven inches.

Concussion, or other external violence, is a frequent cause of partial separation of the placenta, inducing extravasation into the tissue, and frequently giving rise to abortion. That the placenta is the seat of numerous morbid processes may be inferred from the close relation it bears to the nutrition of the fœtus, the frequency of fœtal disease, and the necessary transition through the placenta of any morbific agent, which induces the latter. It is only, however, very recently that the attention of pathologists has been directed to the diseased conditions of this organ ; our knowledge of the morbid changes to which it is liable is, therefore, as yet, very limited.

Professor Simpson* describes congestion of the placenta as affecting the maternal or fœtal portion, causing the external surface of the organ to assume a more or less deep violet, and, sometimes, almost livid colour, the internal structure presenting a deep purple hue, from the vessels being overcharged with blood, while the substance is heavier and more solid than natural. One of the sequelæ of congestion is hæmorrhage into the body, or on the surface of the placenta, varying much in extent. The effused blood undergoes the changes usually traced in coagula, and when there are several fibrinous remains, they cause a tuberculated appearance.

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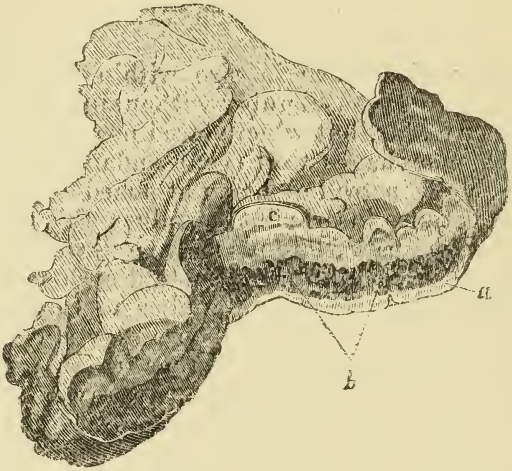
* "Edinburgh Medical and Surgical Journal," vol. xv. p. 265.

Inflammation of the placenta begins from the uterine surface, or in the substance of the organ, and presents the various stages seen in other parts, producing local or general hepatization, effusion, irregular adhesions, and secondary degenerations.

Inflammation may attack the whole, or a portion of the placenta. The effusion and compression of the tissues will vary according to the extent of the inflammation, causing more or less obliteration of the blood-vessels. In an extreme case we find a capsule of dense lymph encasing the maternal surface, the whole is considerably reduced below the normal size, and the soft, spongy texture is converted into a compact splenified mass. This layer must be furnished by inflammation of the membrana decidua. If confined to individual lobules, the alteration will be limited in a corresponding degree. Those instances of adherent placenta which are the source of so much anxiety to the accoucheur, are probably referable to a prior inflammatory attack, glueing the after-birth to the uterus. Professor Simpson admits the occurrence of total absorption of a placenta as one of the consequences which may result from the agglutination of the after-birth to the uterus. A third stage of inflammation is occasionally met with in the shape of abscesses or of purulent infiltration; it is also stated to give rise to the effusion of pus between the two surfaces of the uterus and placenta. Rokitansky describes suppuration occurring here in the form of circumscribed abscesses, or of diffused infiltration and fusion. Fatty degeneration of the placenta, to which Professor Kilian and Dr. Barnes have recently drawn attention, is probably to be explained

Fig. 185.



Sectional view of atrophied placenta. The atrophy and fatty degeneration of the maternal and fœtal portions were caused by a fibrinous layer on the uterine surface. *a*, Fibrinous deposit. *b*, Maternal portion of placenta. *c*, Fœtal portion.

as the molecular disintegration resulting from the deposit of fibrine in the cells surrounding the villi of the chorion; and not as a primary deposit of oil within the placental capillaries, a view more fully developed by Dr. Handfield Jones,* in a paper on fatty degeneration. If portions of a placenta, thus degenerated, be thrown into water, "the first thing which strikes the observer," to employ the words of Dr. Barnes,† is, "that the tufts of villi do not expand or float out in the same way as in the healthy placenta, and on endeavouring to separate the fragments into its component villi with needles, the extreme brittleness of the whole structure becomes apparent." Examined by a high power, "we observe (1) that the villi are thickly studded with innumerable minute spherules of oil; (2) the chorion is much altered; it is thickened and destitute of nuclei; (3) the walls of the vessels no longer contain nuclei, these having, in all probability, become degenerated into spherules of oil; (4) the spherules of oil are contained, some in the chorion, some in the walls of the blood-vessels, and many in the intervals or spaces between these; (5) the cavities of the vessels are almost invariably free from fatty deposition; (6) the vessels are destitute of blood."

"*Uterine Hydatids;*" *Myxoma of the Chorion.*—The villi of the chorion are not unfrequently found to have become converted into oval, pedunculated bodies, like hydatids, clustered together like a bunch of grapes; the cysts vary in size from a pin's head to a filbert, and more, and they may amount to several hundred. This pathological condition has received the name of the vesicular mole; it is to the expulsion of a mass of this kind that the fabulous accounts of women having given birth to several hundred children are attributable; the cysts having been regarded as ova, and these having been magnified into infants. Two beautiful specimens of the disease are preserved in St. George's Hospital Museum.

It appears to depend upon the time when the degeneration occurs whether it shall be total or partial. If the process begin after the first month of pregnancy, it will be confined to the placenta, or even to a part of it. Virchow interprets these structures as myxomatous degeneration, and has shown that the normal villi contain tissue of this kind (mucous tissue), which is continued into them from the umbilical cord.

Rokitansky denies the occurrence of tubercle in the placenta. The only instance on record that we have met with is one described by M. Hardy,‡ as having been found in a phthisical female aged thirty-five; but there seems little doubt, on reading his description, that the supposed tubercles were altered blood-clots, or fibrinous masses.

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* "Med.-Chir. Review," April, 18[illegible]3.
† "Med.-Chir. Trans.," vol. xxxiv.
‡ "Archives Générales de Medecine," 1834, vol. v. p. 244.

THE FŒTUS.

The fœtus is liable to become the seat of morbid processes at every stage of its development and in every tissue and degree; giving rise, at the earlier period of its existence, to an entire destruction of the formative nisus, or to partial arrests of development in individual parts, some of which we have had occasion to allude to in speaking of the malformations of different organs;—inducing in its later intra-uterine existence phenomena of disease resembling those met with in the human being after birth. It remains for future inquirers to determine more accurately, not only the exact pathological character of the different lesions, but also the primary or secondary relation borne between the morbid states of the placenta and the fœtus. Many of the masses that have passed under the name of moles have originated in a blight of the ovum; "the embryo," as Dr. Ashwell describes it, "having died early, the ovum has increased in size and solidity, not by a process of growth, as in natural pregnancy, but by the effusion of coagulable lymph from inflammation of the lining membrane. This forms successive layers over the surface of the dead ovum, giving it, eventually, a great degree of consolidation. Some of these masses exhibit no cavity, but the chorion and the amnion are demonstrable, although the enveloping lymph may be one or two inches in thickness."

Every organ and tissue of the fœtal body may become the seat of atrophy or hypertrophy; the latter may be characterized as actual excess of one or more organs, as we often meet with in the phalanges. Numerous distortions, curvatures, even fractures, and other solutions of continuity,* demand the attention of the medical man immediately after the child's birth. Atrophic conditions are, generally, referable to diseased states of the placenta, which interfere with the nutrition of the child, and cause it to perish, or merely prevent its normal development. Cases are recorded in which such atrophic fœtuses have been borne the full period; though their death has taken place early in pregnancy; these, as Dr. Montgomery† remarks, illustrate the necessity of carefully examining into the state of the fœtal appendages, as to their healthy condition or otherwise, before we venture to pronounce an opinion on the time that has elapsed since conception, merely from the size and general appearance of an ovum or fœtus shown us.

The curvatures that the fœtus is most commonly subject to are, those of the lower extremities—these, as well as the dislocations of the astragalus, the elbow, and other parts that frequently come under the notice of a surgeon, are attributed to violent contractions of the uterus, or to convulsions affecting the fœtus. Of the herniæ to which, as congenital affections, we must allude, umbilical is a frequent and diaphragmatic the rarer form; both are the result of imperfect development of the parietes, which in each case respectively ought to completely close in the abdominal viscera. In the former, the intestines, to a greater or less extent, pass through the umbilical opening, and occupy a pouch formed by the cutaneous coverings of the abdomen; in the latter they enter the thoracic cavity, where they displace the lungs and the heart; they commonly, though not necessarily, cause the death of the fœtus. The brain occasionally protrudes through the cranium, giving rise to hernia cerebri; this, however, must not be confounded with a tumour, which often forms on the head of the infant, simply owing to the mechanical pressure exerted upon it during parturition, and the consequent extravasation, and known as cephalhæmatoma. Encephalocele is described by Dr. Montgomery as, at first, a rather tense, smooth, and semi-transparent tumour, giving generally a more or less distinct sense of fluctuation; in shape the tumour is globular or oval, and frequently tapers to a neck, where it issues from the head, at which point a circular aperture can be detected in the bone, the edges of which are, in general, smoothly rounded off. The defect in the cranial bones, giving rise to this malformation, is analogous to that upon which spina bifida depends; here there is a deficiency in the arches of one or more vertebræ, allowing a protrusion of the dura mater, or sheath of the canal, and the arachnoid lining, in which an accumulation of the spinal fluid takes place. The arachnoid often forms at the most projecting parts of the tumour the only investment, both the skin and the dura mater being thinned down gradually, and at last entirely lost. The fluid, in its turn, presses upon the cord, and more or less displaces it. When it occurs in the lumbar region, its ordinary site, the divided cauda equina may be seen, as Dr. Bright has shown,* adherent to the sac, and induces the erroneous opinion that the appearance is due to the nerves distributed over the sac being turned backwards from their natural direction. When there are several deficiencies in the osseous canal, the fluid communicates between the different tumours; the entire column may be deprived of its spinous processes and their arches, so that the tumour occupies the whole region. Spina bifida is often associated with hydrocephalus.

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* See a remarkable case that was brought before the Medico-Chirurgical Society by Mr. T. D. Jones. "Med.-Chir. Trans.," vol. xxxii. p. 59.

† Art. "Fœtus," in Dr. Todd's "Cyclopdæia of Anatomy and Physiology."

One of the most remarkable occurrences in intra-uterine life is the phenomenon of spontaneous amputation of a limb; this is sometimes complete, the severed extremity being entirely detached, and leaving a stump, in which the healing process is perfected; at others only partial, the stricturing band not having cut through all the tissues. It is generally the left lower extremity that suffers; and Dr. Montgomery has demonstrated the fact of its being due to the umbilical cord being twisted round it, and not, as has been suggested by others, to gangrene, or the accidental formation of ligamentous bands. He expresses his conviction that many of the cases of apparent arrest of development may be set down to this cause, the amputated member not having been found, either from its being atrophied or buried in coagula, and from the separation having been effected at the early stages of pregnancy.

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* "Reports of Medical Cases," vol. ii. p. 640.

Numerous observations are recorded by authors, evidencing the occurrence of the inflammatory process in the fœtal viscera. Peritonitis, with its various sequelæ; gastro-enteritis, followed by ulceration; inflammatory lesions of the liver, pneumonia, and pleurisy; abscesses in the lungs, the thymus, thyroid glands, and supra-renal capsules; and pericarditis; have each been proved to occur in the fœtus, by Desormeaux, Billard, Simpson, Cruveilhier, Montgomery, and other pathologists, to whose works we must refer for further particulars. The same applies to the various cutaneous affections of a syphilitic or other character. Nor is it compatible with our limits to do more than allude to the endless varieties of monstrosities which, from the causes mentioned, or from reasons to which pathology offers no clue, affect the unborn child.

EXTRA-UTERINE PREGNANCY.

Five varieties of extra-uterine pregnancy are assumed to occur—in the Fallopian tubes, in the walls of the uterus, in the ovaries, in the peritoneal cavity, and in the vagina. We have not met with a well-authenticated instance of the last form, and the occurrence of ovarian pregnancy has also been denied by authorities like Velpeau and Kilian. The danger to mother and child is almost equal in each variety; in fact, there is only one case on record in which both have survived; this was one of peritoneal or abdominal pregnancy, which occurred to Dr. P. L. Heim, and in which the Cæsarean section was successfully performed.* The accident most frequently met with is the Fallopian tube pregnancy. In this case the ovum is arrested in its descent into the uterus, and the process of growth and development progresses as if it had reached its proper nidus, up to the period of its discharge. The cause of this stoppage appears to be obstruction in the tube, which is not so great as to prevent the entrance of semen, but does prevent the descent of the ovum. The tube is distended, and its walls become hypertrophied; the changes in the maternal system, though sometimes accompanied by certain anomalous symptoms, are those met in ordinary pregnancy; there is general turgescence of the mammæ and the uterus, and in the latter organ it has long been taught that a decidua is formed, as if it contained the fœtus.

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* Rust's "Magazin für die gesammte Heilkunde," vol. iii. 1817.

Denman, Baillie, William Hunter, and Elliotson, have met with instances in which the decidua was present; other cases have been recorded by Mr. Langstaff and Dr. Lee, in which it had not formed. We have ourselves examined a preparation in St. George's Hospital Museum (No. 2718), in which the decidua is wanting, nor does it seem difficult to understand that in one instance the sympathetic irritation should be set up by which this membrane is produced, and that it should fail in others. The development of the Fallopian tube does not keep pace with that of the ovum, and in the second or third month rupture generally takes place, the fœtus escapes into the abdominal cavity, and the mother sinks from the shock, the hæmorrhage, or the peritoneal inflammation that ensues; but instances are known of the patient surviving, and the fœtus undergoing in the abdomen degeneration to a *lithopædion.* In the majority of instances the right tube is the one affected. Rupture does not appear to be the invariable issue. In the Royal College of Surgeons (preparation No. 2719) we find a fœtus almost completely developed, but compressed and dried, which is stated to have been removed by operation from the Fallopian tube fourteen years after gestation; the patient recovered, and lived for a long time after at Hamburg, where the operation was performed. This specimen is a good instance of what has been termed a lithopædion, a stone-child; the parts intervening between the extremities are ossified, and nutrition appears to have been completely arrested. These lithopædia are sometimes retained within the uterus, as the remarkable preparation in the same museum (No. 2720) proves, of which Dr. Cheston * has given a detailed account. The mother, at the age of twenty-seven, carried her fourth child to the full period, had labour-pains, but no child was born. She recovered, and died paralytic at the age of eighty. The uterus was found to contain an osseous sac adherent to the surrounding part, and resembling a middle-sized human cranium. The cyst seemed to have absorbed all the parts in contact with it, and contained a fœtus in the same position as that in utero. The brain, lungs, and liver preserved almost their natural appearance; but there was no trace of blood, nor any remains of membranes, placenta, or umbilicus. The osseous sac, with the fœtus, weighed three pounds one ounce four drachms. A similar instance, of twenty-eight years' duration, is described by Prael.†

The terminations of Fallopian tube gestations alluded to are not the only issue which we meet with. Adhesions form with different parts of the parietes, and the fœtus having been broken up by ulcerative disjunction, the parts may be discharged piecemeal, whereupon the cyst in which they were contained contracts, and the mother survives. In this way the fœtus has been eliminated by the rectum and the umbilicus.

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* "Med.-Chir. Trans.," vol. v.

† "De Foetu, duo de triginta Annos in Utero detento." Goettingen, 1821.

The foregoing remarks also apply, in the main, to abdominal gestation; here the ovum, probably owing to a want of that erectile tone in the fimbriated extremity of the tube by which, in ordinary pregnancy, it is made to embrace the ovary when conception is effected, falls into the peritoneal cavity. The development proceeds up to a certain point, the fœtus becomes enclosed in very thick and firm membranes, and death ensues from peritonitis, or hæmorrhage, or else the fœtus is eliminated in the manner above described.

Pregnancy in the parietes of the uterus probably consists in an arrest of the ovum at the point at which the tube is inserted into the uterus; the sac, therefore, consists chiefly of the muscular tissue of the uterus, but owing to the irregular development of the organ, the process cannot run its full course to the full period, the walls of the sac in which the ovum lies give way, and hæmorrhage or peritonitis results.

Ovarian pregnancy results from the impregnation of the ovum in the Graafian follicle, where for any reason it is not detached, although the follicle is ruptured. It seems to be an actual occurrence, though extremely rare, and though some authorities have denied the possibility of it.

THE PATHOLOGICAL ANATOMY OF THE JOINTS.

CHAPTER XLIV.

DISEASES OF THE JOINTS.

Malformations.—In cases of defective development, some joints may be quite absent, the bones may be united by congenital anchylosis; or, in a less degree of imperfection, they may be incompletely formed, the ligaments sometimes being partly or altogether wanting, even when the rudimentary extremity of the bone is covered with cartilage. On the other hand, supernumerary joints exist, both when the number of bones is natural and when it is excessive.

Inflammation of the Synovial Membrane.—This may arise as a primary disease spontaneously, from cold, from injuries, from localization of the rheumatic poison, or from that of syphilis, or gonorrhœa. It also occurs as a secondary affection, excited by disease of the cartilage, or of the subjacent osseous tissue. It may be acute in various degrees, or chronic. It is rare in young children, less so about the age of puberty, and very frequent in adults. Before we describe the morbid changes, we must advert to two points in the anatomical arrangement of this membrane, which are of much importance. The synovial are commonly, and no doubt justly, classed with the serous membranes, and are described to form shut sacs, just as these are. Dissection, however, fails to trace the membrane over the free surface of the cartilages, and microscopic examination confirms its absence, except in the fœtus. In these unused joints the cartilaginous surface is found quite smooth and even, and covered by a layer of delicate epithelial scales, such as line the surface of the synovial membrane where it passes over the ligaments. In the articulations, however, of adults, not only is the epithelial layer absent, but the surface of the cartilage is slightly irregular, as if somewhat worn. Our own examinations have convinced us of the general correctness of these statements, given by Dr. Todd and Mr. Bowman, but we must also mention that another high authority, Mr. Toynbee, is of a different opinion, and believes that he can demonstrate the existence of the synovial membrane in the adult, by detaching an exceedingly delicate layer from the cartilage, which, he states, does not contain any of the cartilage cells. This, we think, is a film of the cartilage itself, only so thin that it cannot include the cells. The other point we wish to notice is the existence of a set of remarkable vascular processes, the synovial fringes, covered by a delicate epithelium, upon the free projecting margin of those synovial folds which advance into the cavities of joints. Mr. Rainey, by his discovery of these, has confirmed the anticipation of Clopton Havers, that those synovial folds fulfil, in some measure, the function of glands, being particularly concerned in the formation of the synovia. It is, we are convinced, from these vascular processes that blood-vessels first enter the false membrane formed by exuded lymph.

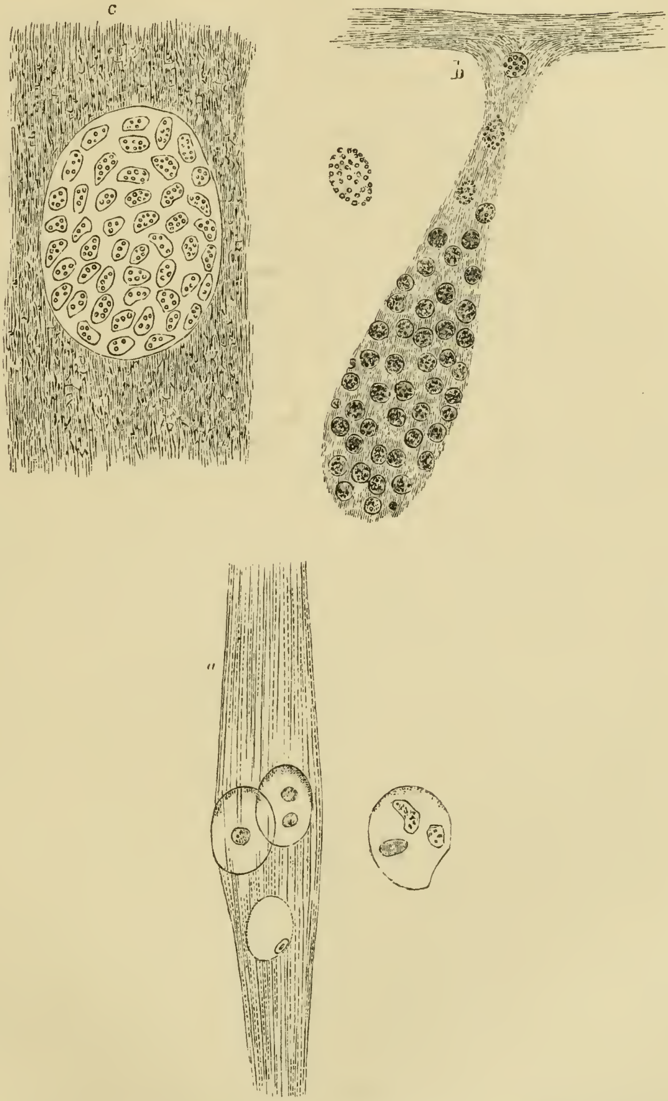
Inflammation of the Joints.—Though this is generally called synovitis, it must be remembered that the cartilages are also concerned.

The results of acute synovitis are the following. More or less injection of the vessels, which in one instance, related by Sir. B. Brodie, were so distended with blood that "throughout the whole of its internal surface, except where it covered the cartilages, the synovial membrane was of a dark red colour, like the conjunctiva in acute ophthalmia." Effusion of serous fluid, which may be so great as to lead one to suppose that the sac is filled with solid matter. Effusion of lymph, forming flakes all over the synovial surface, and not upon the cartilages. In severe cases suppuration may occur. If the disease advance unchecked, ulceration of the cartilages is very prone to occur; villous or fringed processes are then formed, which are in contact with the ulcerating part of the cartilage, and probably both promote the destructive process, and aid in removing, by absorption, the disintegrating tissue. We shall return to this point again under the head of ulceration of cartilage. Under judicious treatment, the whole of the fluid will be reabsorbed, and the joint return to a perfectly healthy state. If, however, much solid exudation is present, its absorption will be more difficult, and some amount of swelling and stiffness of the part will still remain.

Acute Rheumatism.—Though this is perhaps the commonest disease affecting the joints, little is known of its morbid anatomy. In the rare opportunities which occur of examining the joint, it often happens that there is no marked alteration visible to the naked eye beyond slight vascularity. MM. Cornil and Ranvier, however, describe an inflammation of the synovial surface resembling the inflammations of serous surfaces; and marked by the production of large compound cells with several nuclei, corpuscles like those of pus, and sometimes a fibrinous reticulum. They also state that there are constant proliferative changes in the cartilage cells, of the kind hereafter described, resulting in the production of secondary capsules, and splitting up the entire cellular substance.

"Chronic synovitis," Sir B. Brodie says, "causes an increased

Fig. 186.



From a case of secondary deposit in knee-joint, the same as described in the text in the next page.

c, A cartilage cell, immensely hypertrophied, lying in fibrous stuff. *b*, A villous process springing from the synovial membrane. *a*, A strip of fibrous tissue containing three enlarged cartilage cells—one is also figured separately.

secretion of fluid, but does not in general terminate in the effusion of coagulable lymph, or in thickening of the inflamed membrane." Fibrinous matter is, however, if the disease continue long, or often recur, effused either on the inside or outside of the synovial membrane, and becoming gradually organized into a fibroid tissue, thickens its substance and renders it sometimes firm and gristly. A preparation in the museum of St. George's Hospital, shows the synovial membrane of the knee-joint so altered in this way as to be nearly an inch thick. It may be difficult, if not impossible, to detect the presence of fluid in the cavity of a joint which is in this state. Serous effusion, to a considerable amount, sometimes takes place in the synovial sac, without any manifest inflammation. The affection is analogous to hydrocele, and belongs to the class of passive dropsies. Its causes are generally obscure.

Suppuration.—When abscess occurs in a joint, the pus is commonly mixed with more or less of synovial fluid, and flakes of lymph, and is sometimes quite of a sea-green colour. There is, also, sometimes, suppuration outside the joint, the colour of the muscles is altered, the periosteum and the osseous structure in the vicinity are injected and inflamed. A rapid effusion of pus into the synovial cavities of joints, not unfrequently occurs in pyæmia, puerperal fever, erysipelas, and in cases of contamination of the blood by some morbid matter. In one instance of this kind, occurring after a thecal abscess in a finger, we found the synovial membrane forming the margin of one of the ligamenta alaria of the knee-joint manifestly injected, and fringed with a number of various-sized villous projections. These consisted of a fibro-homogeneous, granulous substance, imbedding numerous glomeruli. A layer of similar matter was spread over the whole of the synovial surface, which was not injected with blood. The cartilage was ulcerated in some part of its extent, its surface rendered irregular by superficial erosions, and its texture altered to a lax fibroid stuff. The cartilage cells in these parts were most remarkably changed, containing sometimes from twenty to twenty-five celloid masses in their interior, instead of the two or three which they might contain in their normal state (see Fig. 186); showing, therefore, the same changes as in simple ulceration of cartilage, to be hereafter described. The intercellular substance was entirely deprived of its natural consistence; it broke down under slight pressure. Sometimes complete destruction of the cartilage and part of the bone result from acute suppuration (in pyæmia); but in general the disease has too rapid a course to produce serious changes.

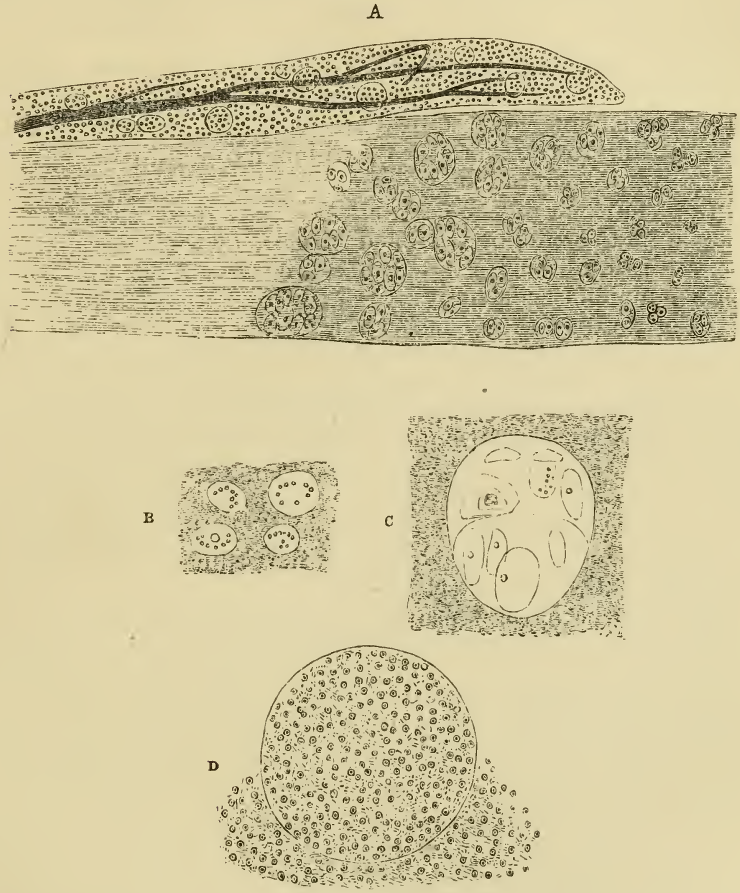
Chronic Suppuration, or White Swelling.—In this condition, according to Rokitansky, "the quantity of purulent fluid effused into the cavity of the joint is generally considerable, and the capsule is, consequently, much enlarged; the synovial membrane is lined with a firm, shreddy layer of lymph, which is dissolving into pus, and a soft, purulent precipitate, which can be easily removed, adheres to the cartilages." The layer of fibrine, lining the synovial membrane, "is opaque and lustreless, its surface is rough, and serum is infiltrated, and blood in small spots extravasated through its tissue, as well as through that of the fibrous capsule of the joint, and neighbouring cellular structures. As the disease advances, the infiltration and thickening of the neighbouring structure increase, they become filled with a gelatinous, lardaceous, white product, in the midst of which fibrous tissues, capsules, ligaments, or aponeuroses, can be no longer recognized. Here and there, in the mass, there are cavities of different dimensions, the lining of which is vascular, spongy, and granulating, and the contents purulent. The muscles near the joint are pale and flabby, infiltered and attenuated. At length the infiltration reaches the subcutaneous cellular and adipose tissues, and the integuments become fixed to the disorganized structures beneath. The diseased joint then presents the following external appearance: it is swollen, and always more or less bent; it feels everywhere soft and flabby, or in some spots flabby, in others firm, elastic, doughy, and at the same time tuberculated; the integuments over it are tense and pallid, leuco-phlegmatic, or they are traversed by varicose veins.

At length ulceration commences, and advances in various directions. "Externally, the capsule ulcerates in one or more spots, and then the soft parts adjoining it. In some instances large openings form in the capsule, and connect the joint with ulcerated cavities in the soft parts; in others, mere sinuses are formed; but in either case they open externally through the skin, and occasion and maintain a discharge of the contents of the joint. Internally, the interarticular cartilages and the ligaments ulcerate, the cartilage covering the bones, when brought into contact with the matter, is destroyed in the way that has been mentioned, and the ulcerative inflammation attacks even the bones, if they have not been involved already. The cavity of the joint appears like a cloaca, surrounded with a gelatino-lardaceous mass; the integuments covering it are of a dark-red hue, and are especially discoloured at the orifices of the sinuses. The joint contains pus or sanies of an offensive odour and variously discoloured, the repeated hæmorrhages which take place when there is acute caries of the bones, very frequently giving it a red or brown tinge; the ligaments ulcerate, and the cartilages separate partly, or entirely, from the bones; the osseous surfaces are laid bare, their compact wall is destroyed, and the spongy tissue is exposed, infiltered with pus and ulcerating, and surrounded on all sides by osteophytes of various shapes; remains of the fibrous structures of the joint, pieces of loosened cartilage, and of necrosed bone, are mixed with the matter discharged from the joint. The soft parts, and the entire bones belonging to the diseased joint, are wasted, most of the fat is absorbed, the muscles are remarkably blanched and thin, and the bones, being generally in a state of eccentric atrophy, are soft and fragile. More or less quickly after the disease has reached this stage, spontaneous dislocations, as they are called, ensue. Chronic suppuration is difficult to distinguish from what is called "scrofulous" disease of the joints, referred to hereafter. Both seem to have been included under the head of "white swelling."

Inflammation may attack the cellular tissue *around* joints, causing effusion of coagulating fluid and consequent swelling, with subsequent formation of pus. One or more spots only may be affected, so that small, local deposits of pus are produced; or the whole may be involved, and the joint become enveloped in a large abscess. In its later stages, the disease extends to the synovial membrane and the cartilages; or recovery may take place, the joint remaining sound.

Pulpy degeneration of the synovial membrane is a very curious and peculiar disease, with the exact nature of which we are yet scarcely acquainted. It was first described by Sir B. Brodie as a morbid alteration of structure peculiar to the articular lining membrane, nothing analogous having been observed in the serous sacs. He says, "The disease seems to commence in the reflected portions of the synovial membrane, converting them into a light brown, pulpy substance, varying from a quarter to a half, or even a whole inch in thickness, intersected with white membranous lines and red spots, formed by small vessels injected with their own blood. It then attacks the synovial membrane of 'the cartilages,' beginning at their edge, and extending gradually over them, ulceration in those cartilages going on correspondingly, till the carious or ulcerating surfaces of the bone are exposed. The cavity of the joint sometimes contains pale yellow fluid in the floating flakes of lymph, or pus, which is discharged externally by ulceration; but sometimes neither. Or abscesses may exist in the altered synovial membrane itself, without communication in the joint." We have given, in the "Pathological Report for 1848-49," a detailed account of the disposition and structure of the synovial membrane thus peculiarly altered, from which we extract the following summary:—The new growth formed prominent fringes of a soft, greyish structure, which overlapped and encroached considerably on the surface of the articular cartilage. The marginal zone of the cartilage, for a varying extent, was converted into a kind of fibrous tissue, and blended with the altered synovial membrane. More internally the cartilage was grooved on the surface, probably so as to correspond with the overlaying fringe. The fibrous tissue into which the cartilage was transformed was of an imperfect kind, not divided into distinct fibres, and not containing any of the natural cells, but strewed over with numerous oil drops and yellowish molecules. The change in the cartilage was effected by extraordinary enlargement of its cells, which were crowded with an endogenous growth of young cells containing each a small oil drop and much clear fluid. At the margin of the cartilage, which was obliquely truncated, the change was most advanced; in the interior the structure was quite natural. The pulpy synovial tissue consisted principally of well-formed nuclei and granular matter, with which were mingled a few fusiform and circular cells. These elements were contained in an enveloping membrane, very thin, of whitish aspect, and nearly homogeneous texture. There existed scarcely any trace of stromal fibres, but a good many large vesicles, or loculi, formed of almost homogeneous walls, and filled with material similar to that which surrounded them. Delicate-walled blood-vessels ramified through the mass, but not in great numbers. It seems very probable that the altered synovial tissue promotes and is concerned in the absorption of the cartilage. The disease almost always occurs before the middle period of life. "In general it can be traced to no evident

FIG. 187.



A, Vertical section of cartilage in process of absorption towards the left, and overlapped by the vascularized pulpy synovial fringe. The edge of the cartilage where it is obliquely truncated is continuous with the fibrous tissue on the left hand.
B, Healthy cartilage cells from the right-hand side, more magnified.
C, Greatly enlarged cartilage cell, containing young cells.
D, Loculus, from thickened synovial membrane, filled and surrounded with nuclei.

cause; but occasionally it is the consequence of repeated attacks of inflammation." It rarely occurs elsewhere than in the knee, but has been seen in the ankle, and in a joint of the fingers. Sir B. Brodie classes it with malignant disease, but from this it is differenced by well-marked characters. It has also been called scrofulous, but Mr. Holmes contends that it is merely chronic inflammation.

Arborescent Growths.—Another very curious alteration, which is sometimes observed in the synovial membrane, consists in its free internal surface being covered by a growth of large villous processes, quite perceptible to the naked eye, which hang into the cavity of the joint, and present a shaggy appearance. They are not developed on the surface of the cartilages. They have sometimes the form of simple threads, or flattened shreds, or their free extremities are split into filaments like a tassel, or they have a club shape, or resemble melon-seeds hanging singly or in clusters from each stalk. In structures they consist of a fibroid material, containing, we believe, in many instances more or less fat, and approaching herein to that peculiar form of fatty tumour which is called *Lipoma arborescens.* The healthy texture of the articulation is not materially interfered with, at least in many cases. These have been often regarded as outgrowths of the synovial fringes, but Virchow has traced some to sessile ecchondroses. Beside the more usual fatty and fibrous structure, some similar bodies contain cartilage, and others bone. Becoming detached from the pedicles, they form some of the masses called loose cartilages.

We have seen both the loose and the pendulous masses in the same joint, in a case of ununited fracture of the neck of the femur, with chronic inflammation. The length of the pedicle varies much, and there seems reason to think that they may be sometimes originally sessile outgrowths (ecchondroses) which become pedunculated. But it seems that cartilage is sometimes found in the normal synovial fringes. In a pendulous body removed by Mr. Shaw from the knee-joint of a girl aged seventeen, the end of a needle was found to be the nucleus of the mass, which consisted of true cartilage and true bone. Mr. Shaw suggested that the needle had probably entered the external condyle of the femur, and there set up an ecchondrosis which became ossified, and finally so far detached as to be pendulous; while evidently it might easily have become completely detached. ("Trans. Path. Soc.," vi. 328.) These pendulous bodies are very common in chronic rheumatoid arthritis.

Loose cartilages, or mures articulares (so-called), are not unfrequent in the cavities of joints. They are usually from the size of a millet-seed to that of a pea, but have been met with as large as a walnut. In shape they are more or less oval and flattened. Their surface is smooth, as if invested by a serous covering, which they sometimes evidently possess, when they are attached to the synovial membrane by a pedicle of varying length. Formations of this kind commence in the sub-serous tissue, and as they enlarge, gradually make their way inward towards the cavity of the joint, in which at last they become free by the dissolution of the pedicle. These are usually true cartilages, and sometimes contain bone, in which may be seen Haversian canals, a clear proof of its having been at one time vascular. Others are formed by a condensation of fibrinous coagula; "they are distinguished," Rokitansky says, "by their uniform smoothness throughout, by a delicate albuminous investing membrane, and frequently by their manifest arrangement in concentric laminæ." These never contain any of the characteristic cells of cartilage, and appear to consist solely of compressed fibrillating exudation. Occasionally they are lodged in ulcerated cavities of the normal cartilage, which might give rise to the idea that they were truly fragments of this tissue, cut out as it were by the process of ulceration. We do not believe, however, that this ever happens. It has also been supposed that fragments of cartilage chipped off may become loose bodies, and the possibility of this occurrence is shown by a case in which Mr. Simon removed from the knee-joint a piece of cartilage thus broken off ("Trans. Path. Soc.," vol. xv. p. 206), but this cannot account for many cases. Calcareous matter is sometimes deposited in the substance of these loose cartilages, and Mr. Rainey describes true bone lacunæ, similar to those seen in the thin plates of the ethmoid, as existing in their interior.

Inflammation of the ligaments, both acute and chronic, is said to occur, but Wickham states that, according to his experience, "the ligaments are the last of all the different parts diseased, and that it is very common to find the ligaments perfect, even when every other texture is either altered or destroyed." From Mr. Key's account it seems that inflamed ligaments become thickened and more pulpy than natural. The areolar tissue which penetrates among their fibres becomes highly vascular, and is probably concerned in producing the softening and ulceration of their substance which sometimes takes place.

Gouty deposit is often found in the ligaments, with or without similar deposit in the cartilages.

Relaxation of the ligaments may be the result of long-continued chronic inflammation, or of simple disuse of the limb. In the latter case it has been known to proceed to such an extent as to allow the head of the femur to slip out of the acetabulum. Frequent and heavy strains may produce a similar effect; Mr. Wickham mentions a case in which the leg was so much bent outward at the knee as to be at nearly right angles with the thigh.

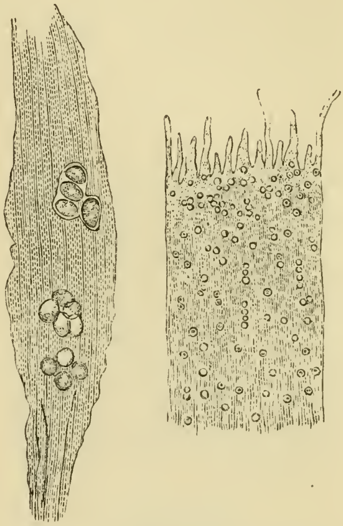
MORBID CONDITIONS OF CARTILAGE.

It is somewhat doubtful whether a true hypertrophy of cartilage ever takes place; but an apparent hypertrophy is not unfrequently observed. The thickness may be increased to treble of that which is normal; at the same time the tissue becomes very soft and yielding, and shows a decided tendency to break up into fibres, which are arranged vertically to the surface. There is some evidence to show that, at a later period, cartilages, so altered, would waste and disappear. In advanced age articular cartilages become considerably thinned—at least this is the case in the hip, and, probably, more or less in all joints that are exposed to pressure. Sometimes the cartilage is simply atrophied; in other cases it is replaced by a semi-translucent, and in others again by a white fibroid tissue. Sometimes the cartilage seems itself to ossify, being converted into what is called the ivory or porcellaneous deposit. This is a peculiarly dense kind of bone; its Haversian canals being filled up by the earthy salts. Besides occurring as a gradual, almost unperceived change in the aged, it is also met with very constantly in the disease termed *chronic rheumatic arthritis*, of which we shall presently speak.

Gouty Deposit in Cartilages.—The free surface of cartilages is sometimes covered with a thin layer of urate of soda; and the same matter may also exist in the substance of the cartilage, in the cancelli of the invested bone, and in the subsynovial tissue. It is deposited as the result of gout. The appearance of the articular surfaces is precisely as if they were smeared with white paint. On examining these in vertical sections it is, however, seen that the greater part, if not all, is really contained in the substance of the cartilage, and that the complete infiltration of the superficial layer causes it to appear as if it were actually deposited externally. The urate of soda is in the form of bunches of crystals, like those shown in Fig. 163, deposited in the intercellular substance.

Atrophy, or Usure of Cartilage.—It is not uncommon, on opening joints which are not apparently diseased, to find the cartilages more or less deficient at one or more points, and this especially in the parts where they have had to bear the greatest pressure. The cartilage is eroded more or less deeply, so that in the seat of the lesion the bone may be exposed, and this with scarce any traces of inflammation in the synovial membrane. Sir B. Brodie "has many times observed a portion of cartilage of a joint wanting, and in its place a thin layer of hard, semi-transparent substance, of a grey colour, and presenting an irregular granulated substance." This indicates a partial atrophy, and destruction of the cartilage, with imperfect replacement of it by a fibrinous exudation. We have examined the knee-joint of a female, aged forty-seven, who died of pleuro-pneumonia, in which the cartilage of the femoral condyles, and of the patella, was manifestly in a state of chronic atrophy, or "usure," as it has been termed by Cruveilhier. There was a sight injection of one of the natural synovial fringes, but no trace of inflammatory action; the joint, externally, appeared quite healthy, and no complaint had been made respecting it during life. The cartilage of the patella was most affected; it presented, at its external part, an unequal, irregular surface, about the size of a fourpenny piece, which was softened in texture, and roughened by small greyish prominences. The surrounding cartilage was in a commencing state of similar change. In a vertical section of the part most affected, it was seen that the cells near the free margin were enlarged and multiplied, while the matrix at the margin broke up into fibres of various size, quite separate from each other, the larger still imbedding some of the cells. The accompanying cut illustrates this degeneration of cartilage.

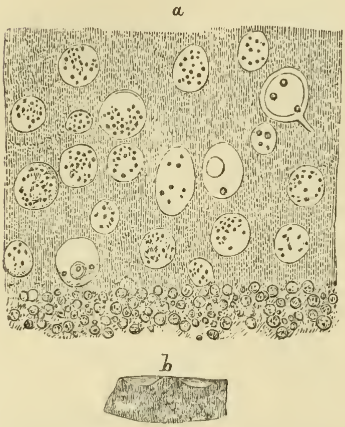
FIG. 188.



Vertical section of cartilage of patella, in state of usure. The free margin presents a number of fibres. The left-hand figure represents one of these fibres, more magnified, and containing some groups of corpuscles.

Ulceration of cartilage, occurring as an acute or subacute affection, in various articular inflammations, has been much inquired into, and our knowledge respecting it has become tolerably definite and exact. It was formerly much disputed whether the change was effected by the action of the vessels of the cartilage itself, or those of the synovial membrane on the encrusted bone. Now, however, we know certainly, that human articular cartilage is entirely devoid of vessels; and we have good reason to believe, that those of the surrounding textures are not the effective agents in the ulcerative process. If we refer, as we may fairly do, to the instance of the cornea, as a very analogous tissue, we can scarcely hesitate to admit that ulceration is essentially an alteration of the nutrition of the affected texture, and that the influence of the adjoining vessels upon it is only secondary. The perforating ulcer of the stomach is also a striking instance of the truth of this position. It being then admitted, that ulceration of cartilage is produced by a special disorder of its own nutrition, we proceed to inquire what has been ascertained respecting the nature and the stages of this diseased action. These were admirably set forth by Mr. John Goodsir, in his well-known paper on the process of ulceration in articular cartilage, an extract from which we subjoin: "If a thin section, at right angles, be made through the articular cartilage of a joint, at any part where it is covered by a gelatinous membrane in scrofulous disease, or by false membrane in simple inflammatory condition of the joint, and if this section be examined, it will be found to present the following appearances on one edge of the section is the cartilage unaltered, with its corpuscles natural in position and size; on the opposite edge is the gelatinous or false membrane, both consisting essentially of nucleated particles, intermixed, especially in the latter, with fibres and blood-vessels; and, in the former, with tubercular granular matter. In the immediate vicinity, and on both sides of the irregular edge of the section of cartilage, where it is connected to the membrane, certain remarkable appearances are seen. These consist, on the side of the cartilage, of a change in the shape and size of the cartilage corpuscles. Instead of being of their usual form, they are larger, rounded, or oviform; and instead of two or three nucleated cells in their interior, contain a mass of them. At the very edge of the ulcerated cartilage, the cellular contents of the enlarged cartilage corpuscles communicate with the diseased membrane by openings more or less extended. Some of the ovoidal masses in the enlarged corpuscles may be seen half released from their cavities by the removal of the cartilage; and others of them may be observed in the substance of the false membrane, close to the cartilage, where they have been left by the entire removal of the cartilage which originally surrounded them. If a portion of the false membrane be gradually torn off the cartilage, the latter will appear rough and honeycombed. Into each depression on its surface, a nipple-like projection of the false membrane penetrates. The cavities of the enlarged corpuscles of the cartilage open on the ulcerated surface by orifices of a size proportional to the extent of absorption of the walls of the corpuscles, and of the free surface of the cartilage. The texture of the cartilage does not exhibit, during the progress of the ulceration, any trace of vascularity. The false membrane is vascular, and loops of capillary vessels dip into the substance of the nipple-like projections, which fill the depressions on the ulcerated surface of the cartilage (Fig. 189); but, with the exception of the enlargement of the corpuscles, and the peculiar development of their contents, no change has occurred in it. A layer of nucleated particles always exists between the loops of capillaries and the ulcerated surface. The cartilage, where it is not covered by the false membrane, is unchanged in structure. The membrane generally adheres with some firmness to the ulcerating surface; in other instances, it is loosely applied to it; but in all, the latter is accurately moulded to the former. In scrofulous disease of the cancellated texture of the heads of bones, or in cases where the joint only is affected, but to the extent of total destruction of the cartilage, over part or the whole of its extent, the latter

Fig. 189.



Drawing of ulceration of cartilage.

a, Vertical section of ulcerating cartilage magnified. *b*, Naked-eye view, showing two ulcerated depressions.

is, during the progress of the ulceration, attacked from its attached surface. Nipple-shaped processes of vascular cellular texture pass from the bone into the attached surface of the cartilage; the latter undergoing the change already described. The processes from the two surfaces may thus meet half way in the substance of the cartilage, or they may pass from the attached surface, and project through a sound portion of the external surface of the cartilage, like little vascular nipples or granulations. The cartilage may be thus riddled, or it may be broken up into scales of various size and thickness, or it may be undermined for a greater or less extent, or be thrown into the fluid of the cavity of the joint in small detached portions, or it may entirely disappear." Mr. Goodsir believed the cells of new formation, the nucleated particles of the false membrane, to be the immediate agents in the absorption of the cartilage. We have had several opportunities of verifying the above account, and have already mentioned two instances in which we observed a similar change in ulcerating or wasting cartilage.

Proliferation of Cartilage Cells.—The description given by Goodsir, many years ago, has been confirmed, not only by the observations in the text, but by those of Redfern and others. Of late years it has assumed much importance as the type of an inflammatory process leading to suppuration in non-vascular parts, and comparable in this respect to inflammation of the cornea. Numerous observations on inflammation of cartilage, produced artificially, have been made by Kremiansky, Cornil and Ranvier, and others. The general result has been to show that inflammation produces the same series of changes described in the text; the cartilage corpuscles breaking up into groups of young cells not to be distinguished from pus corpuscles; as in the cornea (p. 88). Cohnheim, regarding all pus corpuscles as proceding from the vessels, contends the multiplication of the cartilage (or corneal) cells is only a process of breaking-up, or retrograde, not progressive metamorphosis; and that the pus corpuscles actually observed all come from the vessels. It is generally admitted that the number of corpuscles thus produced by the cartilage cells must be small, and probably the proliferative process soon ceases. Heitzmann,* in numerous careful observations on cartilages injured by heat, could not trace any actual pus-formation. In some of the affections in which proliferation occurs, as chronic rheumatoid arthritis, and acute rheumatism, MM. Cornil and Ranvier distinguish the new cells from pus, or inflammation cells, regarding them as actual cartilage capsules of secondary formation; and state that the wall of the cartilage capsule can always be distinguished by iodine. In purulent synovitis they believe actual pus to be produced.

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* Stricker's "Mediz. Jahrbücher," 1872, part iv.

No doubt can exist of the propriety of giving a distinctive name to this affection, although its course does not seem to be exactly similar to that of scrofulous disease of other parts. The articular extremities of the bones are the primary seat of mischief, they become preternaturally vascular and much softened, so that they are easily cut with a knife, while "a transparent and afterwards a yellow cheesy substance is produced in their cancelli." From the observation above mentioned, we are inclined to think that granular cells, formed in the primary exudation within the cancelli, play some part in the absorption and removal of the earthy salts of the bone. As the disease of the bone advances, ulceration of the cartilage commences on its attached surface, in the manner described in the extract we have given from Mr. Goodsir's paper. Before, however, this can take place to any great extent, the articular lamina, so well investigated by Mr. Birkett, must be removed. This consists of a thin lamina of dense bone, containing large lacunæ, with scarce any canaliculi, which bounds and closes in the cancelli on the surface encrusted by the cartilage. Until this is removed no vessels can shoot into the nipple-shaped processes of false membrane which dip into the cartilage. The osseous tissue gradually wastes and is absorbed, it undergoes a true caries; sometimes also a part dies, and may exfoliate. In cases of this kind it is not at all unfrequent to find several joints affected with the same morbid change in various stages. As the whole of the articulating surface is generally involved in the disease, the attachment of the cartilage becomes loosened at all points, and it is, therefore, even at an early period much more easily detached from the bone than is natural. Sometimes, as Sir B. Brodie mentions, in the advanced steps of the disease, nearly the whole of the cartilage is found forming an exfoliation instead of being ulcerated. "As the caries of the bones advances, inflammation takes place of the cellular membrane external to the joint. Serum and afterwards coagulated lymph is effused; and hence arises a pulpy and elastic swelling in the early, and an œdematous swelling in the advanced stage of the disease, which is one form of white swelling. Abscess having formed in the joint, it makes its way by ulceration through the ligaments and the synovial membrane, and afterwards bursts externally, having caused the formation of numerous and circuitous sinuses in the neighbouring soft parts." This disease of the joint especially affects children; it is the essence of the *morbus coxarius* which is so very common among the offspring of the poorer classes. It rarely occurs after the age of thirty. This disease is not regarded by all pathologists as originating in the bone; some regard it as primarily a disease of the synovial membrane. For instance, Billroth describes the affection as "fungous arthritis," and defines it as essentially depending upon excessive growth of the synovial membrane and its fringes. In early stages the lateral portions are found especially altered; the fringes swollen and thick; the whole membrane soft and succulent. Gradually these changes become more marked, the synovial membrane thicker, more œdematous, and red, while the fringes become masses resembling spongy granulations. These begin to grow over the cartilage from the sides, insinuating themselves between the articular surfaces, which they gradually cover completely. The red granulation mass thus formed adheres to the cartilaginous surface here and there by vascular processes, which dip down into the cartilage (much as described by Goodsir.) By the gradual extension and penetration of these the cartilage is destroyed, and the granulating mass comes in contact with the bone. In the meantime the cartilage-cells undergo active proliferation, and the intercellular substance becomes absorbed. The amount of suppuration varies much, and the subsequent destruction of the joint and the ends of the bones, is attributed by Billroth chiefly to the growth of the granulation tissue.

Rindfleisch regards the disease as essentially one of the articular ends of the bones; *caries fungosa*, as he calls it, which attacks and destroys the cartilage beneath, as the synovial outgrowth does from above.

Cornil and Ranvier, on the other hand, contend that the granulation-tissue and fungoid growth are merely secondary inflammations, not peculiar to this disease, the essential feature of which they believe to be fatty degeneration of the cartilage. The latter constitutes the first stage of the disease; in the second stage appear the fungous granulations.

TUBERCULAR DISEASE.

MM. Cornil and Ranvier describe a strictly tubercular disease of the synovial membranes, in which they are thickened, soft, and contain numerous miliary tubercular granulations. The cartilages are affected as in the early stage of scrofulous disease; but the bones are not altered. This type of disease is probably rare. Its symptoms and history are hardly known.

DISEASE OF THE SPINAL COLUMN.

The joints of the vertebræ are very liable to be affected in nearly the same way as other articulations of more perfect development, so that a brief account of their morbid states will properly follow here. The cancellous tissue may be the seat of the scrofulous disease just described, occasioning caries and deposition of cheesy matter in the cavities. "In these cases ulceration may begin on any part of the surface, or even in the centre of the bone; but in general the first effects of it are perceptible where the intervertebral cartilage is connected with it, and in the intervertebral cartilage itself. In other cases the vertebræ retain their natural texture and hardness, and the first indication of the disease is ulceration of one or more of the intervertebral cartilages, and of the surfaces of bone with which they are connected." These cases may be considered analogous to those of primary ulceration of the cartilages of diarthrodial joints. "There is still another order of cases, but these are of more rare occurrence, in which the bodies of the vertebræ are affected with chronic inflammation, of which ulceration of the intervertebral cartilages is the consequence." To this we have not any analogue among the more ordinary diseases of joints, except in the scrofulous disease of joints just described. "In whichever of these ways the disease begins, if not checked in its progress, it proceeds to the destruction of the bodies of the vertebræ and intervertebral cartilages, leaving the posterior parts of the vertebræ unaffected by it; the necessary consequence of which is an incurvation of the spine forward, and a projection of the spinous processes posteriorly." The chronic inflammation of the bones sometimes extends to the membranes of the spinal cord, and when the curvature is very great the cord may be so compressed that it cannot properly discharge its functions. "Suppuration sometimes takes place at a very early period; at other times not until the disease has made considerable progress. The soft parts in the neighbourhood of the abscess become thickened and consolidated, forming a thick capsule, in which the abscess is sometimes retained for several successive years; but from which it ultimately makes its way to the surface, presenting itself in one or another situation, according to circumstances. In the advanced stages of the disease, new bone is often deposited in irregular masses on the surface of the bodies of the neighbouring vertebræ; and where recovery takes place, the carious surface of the vertebræ above coming in contact with that of the vertebræ below, they become united with each other, at first by soft substance, afterwards by bony ankylosis. The disposition to ankylosis is not the same under all circumstances; it is much less where the bones are affected by scrofula than where they retain their natural texture and hardness; and this explains, wherefore, in the former class of cases, a cure is effected with more difficulty than in the the latter. Occasionally portions of the ulcerated or carious bone lose their vitality, and having become detached are found lying loose in the cavity of the abscess." When a large abscess has formed the pressure of the matter on the surfaces of the contiguous vertebræ may cause an extensive caries far beyond the limits of the original disease.

ANKYLOSIS.

The term is derived from the Greek αγκυλη, which signifies a curve of a joint, and was applied to such cases of stiff joint as remained fixed in a curve, not a straight position, which was distinguished by the term ορθοκωλον. The process which produces ankylosis is very similar to that which unites the two ends of a fractured bone, and the union in both cases may be effected either by soft fibroid tissue, or by actual bone. For the production of ankylosis it is essential that the cartilage encrusting the articular bony surfaces should be wholly or in great part removed. When this is effected, and the conditions of the inflamed parts are such as do not tend to the production of pus, but of plastic exudation, the capillaries, which have entered the processes of false membrane from the bony surfaces, meet and anastomose together; so that the vascular systems of the two bones are in free communication, while the intervening fibrinous exudation gradually undergoes change into a dense fibroid tissue, mingled, as Rokitansky says, with an abundance of fat. If the process stop at this point, soft or fibrous ankylosis is the result. More commonly, however, under favourable conditions of perfect rest, proper food, and attention to the health, the exudation undergoes change into osseous substance, so that the two bones become as it were accurately welded together.

False Ankylosis.—There is a kind of ankylosis which is denominated the *spurious* or *false*, most common after synovitis, and which depends on the presence of masses of exudation within the synovial capsule, with thickening of this membrane and of the ligaments. The condition of the muscles also seems to promote the fixed state of the articulation, the extensors being paralyzed and wasted, and the flexors, which exert in most instances a superior power, being contracted, shortened, and atrophied. Ankylosis of the bodies of the vertebræ has already been alluded to as the mode in which a cure takes place after caries of their structure and ulceration of the intervertebral ligaments. It is clear from this that when the destruction of these parts has been at all extensive, the avoidance of the deformity of angular curvature is impossible. Ankylosis of some of the less important joints occurs almost naturally in old age. Some rare instances are recorded in which all the joints of the body became spontaneously ankylosed.

CHRONIC RHEUMATOID ARTHRITIS.

The chronic inflammatory nature of this affection is extremely well marked, but the essential dependence of it upon rheumatism is not demonstrated; in fact, though in an acute form it has a great resemblance to acute rheumatism, it appears to be essentially a different disease. It occurs not only after an attack of acute rheumatism, but after injuries and bruises, and sometimes without apparent cause. It is very frequent in the hip, the shoulder, the knee, and the articulations of the hand. When it is fully established in the hip joint, it is said by Mr. R. Adams rarely or never to extend itself to the other articulations. Sometimes both hips only are attacked. When the knee is the seat of the disease, or the shoulder, other joints will, generally, be found more or less implicated. In the case of the knee, Mr. R. Adams recognizes a first stage, "marked by evidences of sub-acute inflammation, such as pain, heat, considerable swelling. This is followed by a second period, in which the heat and swelling diminish, but the pain continues." We quote from Mr. W. Adams's communication to the Pathological Society, the following account of the appearances ordinarily observed in the advanced stages of chronic rheumatoid arthritis.

"*In the hip joint:* 1st. Great enlargement and irregularity of shape of the head of the femur, which assumes a mushroom-like form, in consequence of real or apparent flattening of its upper part, and nodulated masses and flattened ring-like layers of new bone, surrounding the edge of its articular cartilage, and extending to a variable distance over its articular surface. To this mushroom-like form, the apparent shortening of the neck, in consequence of its upper part being concealed by the overhanging margin of new bone at the edge of the articular cartilage, also contributes. 2ndly. Absence of articular cartilage to a greater or less extent, and the eburnation of the bony surface. 3rdly. Nodulated masses of new bone, from the size of a hemp-seed to that of a walnut, attached by thin peduncles to the synovial membrane on the neck of the bone, or to that of the capsular ligament, —more or less spherical when small, but flattened and irregular when of large size. *In the os innominatum:* 1st. Increased capacity of acetabulum. 2ndly. Ossification of the fibro-cartilaginous rim, or cotyloid ligament. 3rdly. Absence of articular cartilage to a greater or lesser extent, and eburnation of the exposed bony surface. 4thly. Irregular osseous growths (stalactitic osteophytes) on the surface of the bones external to, and immediately surrounding, the joint. *In the knee-joint*, the appearances were essentially similar to those in the hip; new osseous growths, of irregular form, surrounded the margins of the articular cartilages of the femur and tibia; and pedunculated osseous growths, in considerable numbers, and of all sizes, were attached to the synovial membrane, both in the notch and lining the capsule. In addition, however, the articular cartilages on the condyles of the femur presented a thickened nodulated appearance in their central parts." Mr. R. Adams, describing the condition of the shoulder-joint, says, "The capsular ligament is occasionally increased in thickness, and its fibres are hypertrophied; and it is generally more capacious than natural, showing that effusion of synovia to a considerable amount had existed, although the external signs of this phenomenon are not usually evident. When the interior of the synovial sac is examined, it will be found to present evidences of having been the seat of chronic inflammation. Bunches of long organized fringes hang into the interior of the synovial sac; and many of these vascular fimbriæ, which in the recent state are of an extremely red colour, surround the corona of the head of the humerus. We also notice rounded cartilaginous productions, appended by means of membranous threads attached to the interior of the various structures which compose the joint." The size and shape of these bodies are various. The long tendon of the biceps muscle is very commonly adherent to the superior extremity of the bicipital groove, while that portion of it which normally passes upwards, and takes its attachment to the upper margin of the glenoid cavity, is destroyed. The articular surface of the humerus is very much enlarged, and extends itself over the greater and lesser tuberosities, and even over the highest part of the bicipital groove. The head appears to be in a line with the shaft of the bone, instead of being directed upwards, inwards, and backwards. The cartilage is more or less completely removed, the bone in some parts eburnated, in others porous. Nodules of bone, vegetations, as Mr. R. Adams terms them, are thrown out around the margin of the head. The glenoid cavity of the scapula becomes much enlarged, and losing its oval shape assumes a more circular form. This, however, depends much on the position which the head of the humerus occupies. The depth of the articular cavity is increased by osseous productions thrown out around its margins; its encrusting cartilage is removed, and the surface in part is covered by porcellaneous deposit, in parts remains porous. The enlarged head of the humerus comes into immediate contact, in many cases, with the under surface of the coraco-acromial vault, causing absorption and wasting of the tendons of the supra-spinatus and biceps, and the upper part of the capsular ligament. The acromion process, the outer extremity of the clavicle, and the coracoid process, in most cases become enlarged, though their under surfaces are worn and eburnated by the friction and pressure of the head of the humerus. Occasionally, however, they are found atrophied, or altogether removed. It is a remarkable circumstance, particularly noticed by Mr. R. Adams,—from whose article, in the "Cyclop. of Anat. and Phys.," we have taken the foregoing account,—that, in many cases the acromion process is traversed in the line of junction of its epiphysis, "by a complete interruption of its continuity, as if fractured." This has been considered by several observers, as well as the destruction of the long tendon of the biceps, to be the result of accidental violence. Cruveilhier is quoted by Mr. R. Adams as describing the bones of the carpus, in a case of chronic rheumatic arthritis of the wrist-joint, to be so confounded together into an irregular mass that it was difficult to say which part each took in the construction of the carpal region. The radius and the ulna undergo like changes to those which have just been described; the lower surface of the latter, confronting the cuneiform bone, becomes smooth and polished, the inter-articular fibro-cartilage having been removed.

Minute Changes in Chronic Rheumatoid Arthritis.—The changes in this disease seem to depend mainly upon changes in the cartilage, partly atrophic, partly hyperplastic. Considerable absorption of cartilage takes place, and this is described by Rindfleisch as resulting from a process such as we have called above atrophy, or usure of cartilage; consisting essentially in proliferation of the cartilage cells and fibrous change, followed by softening of the intercellular substance. This goes on in the more central parts of the cartilage, where they are exposed to most pressure. The simultaneous hyperplastic changes which take place in the peripheral parts were admirably described many years ago by Mr. W. Adams; * who showed that they consisted in (1) "Hypertrophy of the articular cartilage, generally occurring at the circumferential margin, but occasionally taking place towards the central parts of the articular surfaces. The new growth of cartilage takes place principally, if not entirely, near to the articular surface." It is very similar, though not quite identical with the original cartilage; a fibrillated character of the matrix, and the scattered, solitary, or imperfectly-grouped arrangement of the nuclei being the principal points of difference. Rindfleisch describes it as that species of cartilage which is about to undergo ossification. (2) "In the development of true osseous tissue in the hypertrophied cartilage, ossification commencing either in the newly-formed cartilage, or at the junction of the new with the old cartilage. Ossification proceeds more rapidly in the newly-formed and forming cartilage, (the growth of which is probably simultaneous with the advancing ossification) than in the old articular cartilage: so that considerable masses of new bone are formed, altering the configuration of the articular extremities, whilst a layer of articular cartilage remains in its normal position. More slowly, but as perfectly, ossification takes place in this imbedded layer of articular cartilage. The process resembles the normal process of ossification in temporary cartilage in the intercellular matrix being the primary seat of earthy impregnation, and in the enlargement of the cells in the immediate vicinity of the bone." Although the opposed surfaces of bone become worn away, no caries ensues, the surface being protected by indurative ostitis, which produces the well-known hard ivory-like investment. The pendulous bodies which surround the head of the bone are also, no doubt, in part, originally ecchondroses.

Effects of Dislocations.—The most common cause of dislocations is a violent strain or injury to the part; but they may also come to pass spontaneously, either from abnormal relaxation of the ligaments, or from destruction of them in consequence of disease and muscular inaction. Dislocation, it is affirmed, may also occur congenitally. What we wish to notice here is, the changes which take place in the articulating surfaces, when a dislocation has taken place, and remained a long time unreduced. Rokitansky describes these as follows: "The capsule becomes enlarged, and the place of its insertion altered; the articular cavities of the bones increase in size, and undergo various changes in form; and corresponding alterations are produced in the articular heads or prominences. In other cases, in which the dislocation is complete, the capsule wastes, and the bony cavities diminish in size, or are filled with masses of new osseous substance; the displaced head of the bone loses its character, and a new joint is formed. The cellular structures which surround the dislocated head inflame, and frame a new capsule around it, which, for the most part, fits closely, is of fibroid structure, and has a serous lining; whilst the pressure of the head, in its new position, occasions a shallow, articular excavation beneath it. In other cases, instead of an excavation beneath the head, a mass of callus springs up around it, and forms either a hollow to receive it, or a level surface, while the head may be flattened in order to fit; or, lastly, the callus may project, and that which was the articular head be excavated to receive it. Sometimes the quantity of new bone deposited around a dislocated head is very abundant, and retains it firmly in its place. In dislocations of long standing, the pressure upon the vessels and nerves interferes with the nutrition of the luxated bone, and, like the soft parts, it is found in a state of atrophy."

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* "Report of the Pathological Society," 1850-51, vol. iii. p. 156.

Morbid Conditions of Bursæ.—These small synovial sacs are liable to be affected much in the same way as larger. They may be attacked by inflammation, more or less acute, or quite chronic, resulting from rheumatism, the abuse of mercury, or some other constitutional affection, or excited by violence, or long-continued pressure. The effusion which takes place may, in cases of a chronic kind, be a simple synovial or serous fluid; but when the inflammation is more acute, it is either a turbid serum, with flakes of fibrinous matter floating in it, or actual pus. Suppuration sometimes is produced artificially, for the purpose of causing the obliteration of the cavity of the bursa. The matter sometimes makes its way directly to the surface of the skin, and is discharged, but it often, also, escapes into the surrounding cellular tissue, and diffuses itself over a considerable extent. Sir B. Brodie describes this as being of common occurrence after inflammation of the bursa patellæ, so that an abscess is formed between the skin and the fascia, covering the whole of the anterior part of the knee, and liable to be confounded with inflammation of the synovial membrane of the joint. When severe inflammation supervenes, after the puncture of a large bursa, so much constitutional disturbance is sometimes occasioned that the patient dies.

This is more likely to occur in persons who are in a bad state of health. The walls of an inflamed bursa sometimes become prodigiously thickened by the organization of layers of fibrinous effusion. There is a specimen in the museum of St. George's, in which the walls of an enlarged bursa patellæ are more than half an inch thick, while the cavity, which is comparatively small, is traversed by reticulating laminæ of false membrane. "When the inflammation has been of long standing," Sir B. Brodie says, "it is not unusual to find, floating in the fluid of the bursa, a number of loose bodies, of a flattened oval form, of a light brown colour, with smooth surfaces, resembling small melon-seeds in appearance. There seems to be no doubt that these loose bodies have their origin in the coagulated lymph, which was effused in the early stage of the disease; and I have had opportunities, by the examination of several cases, to trace the steps of their gradual formation. At first, the coagulated lymph forms irregular masses, of no determined shape, which afterwards, by the motion and pressure of the contiguous parts, are broken down into smaller portions. These, by degrees, become of a regular form, and assume a firmer consistence, and, at last, they terminate in the flat oval bodies which have been just described." They are, therefore, like one class of loose bodies in joints. The synovial sheaths surrounding the flexor tendons of the fingers, as they pass under the annular ligament, are not unfrequently the seat of increased secretion of fluid, and of the formation of small bodies, compared by Mr. R. Adams to grains of boiled rice. "They are found in vast numbers in the same cyst, mixed with a more or less considerable quantity of glairy synovial liquid." They occasion, as they move to and fro, a distinct sensation of *frottement*, and are quite identical with those described by Sir B. Brodie.

What are called *ganglions* are small collections of fluids in bursal cavities of new formation. They are most frequent on the back of the wrist and forearm. They do not seem to arise from inflammation, but rather to be of the nature of simple cysts. They are slightly movable, indolent, and painless, and appear to be situated "in the reticular tissue, which immediately covers the sheath of the extensor tendons." To the latter they are connected firmly, to the skin but loosely. The consistence of their contents varies from that of limpidity to that of thick jelly-like matter.* According to Velpeau, these cysts occasionally communicate with the articular synovial cavity.

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* Some fluid of this kind, which we examined, was coagulated, in great measure at least, by nitric acid; it contained a few nucleated granulous corpuscles.

THE PATHOLOGICAL ANATOMY OF THE OSSEOUS SYSTEM.

CHAPTER XLV.

Congenital Malformations.—There may be (1) deficiencies, (2) excessive development, (3) imperfect union of parts, (4) fusion of separate parts, (5) too early or too late development.

1. Defective formation is seen in imperfectly-formed monsters: in the occasional deficiency of a limb or limbs, of fingers, of vertebræ, or other single bones. 2. Excessive development in the occasional monstrous growth of particular bones or limbs, and in the production of supernumerary fingers or other parts. There must, of course, in both these cases, be some corresponding deficiency or excess of the soft parts. 3. Imperfect union of parts is seen in such deformities as cleft palate and spina bifida. 4. Fusion of separate parts is sometimes seen in the union of bones of two fingers or toes into one, or of the bones of the skull. 5. Premature development occurs sometimes in the cranial bones and in those of the limbs, causing too early union of the sutures, and premature union of epiphyses; retarded development is seen in the late closing of the fontanelles of the skull, and the late union of epiphyses in the long bones. Premature synostosis of the cranial sutures is commonly regarded as a cause of small development of the head, or "microcephalism." Dr. Langdon Down, however, after examining 200 microcephalic skulls, contends that the sole cause is imperfect development of the brain, the sutures being generally unossified.

INFLAMMATION OF BONE.

This does not essentially differ from inflammations of soft parts, though, of course, the naked-eye appearance presents certain peculiarities. It is usual to classify the varieties of inflammation according to the part of bone affected—as ostitis, osteomyelitis, and periostitis; the first denoting inflammation of the compact bony tissue; the second specially referring to the medulla and cancellous parts of bone; the last not only inflammation of the periosteum, but of what is practically almost inseparable from this, the contiguous part of the bone. Moreover, in each part of bone the inflammation may take one of two types; it is either *plastic*, which in this case means tending to ossification and induration (sclerosis); or else *destructive*, that is generally suppurative (abscess); but each of these passes through the same preliminary stage, now to be described.

Rarefactive Inflammation.—This variety or stage of inflammation is marked by absorption or rarefaction of the bone. Most pathologists regard the process of rarefaction as the necessary preliminary to either of the others. It appears to be strictly analogous to the physiological process by which the medullary cavity is hollowed out, and spongy, cancellated bone formed. The first appreciable inflammatory changes in bone, to use Mr. Goodsir's words, "occur within the Haversian canals; these passages dilate, or become opened up, as may be seen on the surface of an inflamed bone, or better in a section. The result of this enlargement of the canals is the conversion of contiguous canals into one cavity, and the consequent removal or absorption of the osseous texture of the part." The whole tissue, consequently, becomes much lighter and more porous. The spaces formed by the enlarged canals, as also those formed by absorption of the intermediate material, become filled with granular tissue, composed chiefly of embryonic cells. The further course of the inflammation depends upon whether these cells become formative cells (osteoblasts), or whether they degenerate into pus cells.

The naked-eye appearance of the bone in this condition is thus described by Mr. Holmes:* "When the periosteum is stripped off, the bone is seen to be irregularly vascular, some portions having a slightly red tint, evidently caused by the enlargement of the vessels which pass from the periosteum into the bone; and, with a magnifying-glass, the increased size of the vascular apertures is easily proved, while, if pressure be made on the surface of the bone, drops of blood will frequently ooze from the mouths of the enlarged vessels, proving their increase in size, as well as the loss of consistence in the outer hard wall of the bone." Less obvious changes are perceived in the central parts of the bone. The spaces produced by absorption of the walls of the Haversian canals at length, however, become visible to the naked eye, and spaces are seen in the compact tissue like those in cancellous tissue, so that it is sometimes said to become cancellous. Similar changes occur in the cancellous tissue itself, whereby its cells become enlarged, and sometimes the whole bone becomes expanded by the simultaneous yielding of its walls, and sometimes so much softened as to crackle under pressure (Holmes). A bone in this condition, if macerated, shows a certain degree of osteoporosis.

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* "System of Surgery," 2nd edition, 1870, vol. iii. p. 733.

Sequelæ of Rarefactive Inflammation.—The process just described may, it is probable, come to an end, and resolution take place, without further consequences. In such a case the bone does not, according to Förster, remain in the osteoporotic condition, but gradually becomes filled up by the sclerotic process, hereafter spoken of. A more pronounced form of rarefactive inflammation, called fungous ostitis by MM. Cornil and Ranvier, must be very rare. In general, the rarefactive process is only the first stage of a more enduring morbid process; and since the future course of the inflammation differs according to the part affected, we must describe separately the periosteal, osteal and osteomyelitic affections.

The different forms and results of inflammation of bone are thus summarized by Mr. Holmes:—The products of ostitis are divisible into two principal varieties; on the one hand the deposition of earthy matter and formation of new bone; on the other hand suppuration. The former result terminates in hardening, or sclerosis, the latter in a variety of conditions. When the suppuration is limited within a cavity, circumscribed abscesses are produced; when the pus extends along the inner surface of the membrane lining the medullary cavity and cancelli, it produces the condition now known as osteomyelitis, formerly called "diffused suppuration in bone;" suppuration between the periosteum and bone forms *periosteal abscess*, acute or chronic; and any of these forms of suppuration, when accompanied by the insensible exfoliation of the bone, constitutes ulceration of the bone, or *caries*. Lastly, inflammation of the bone sometimes leads to the death of larger portions of its tissue; this constitutes gangrene of bone, or *necrosis*, which, however, like other forms of gangrene, is often produced by other causes not inflammatory.

PERIOSTITIS.

Acute inflammation of the membrane is characterized by a red blush, a humid succulent appearance, with much swelling and thickening of the membrane, and more or less of a serous effusion, causing a slight separation from the bone. The periosteum may, as Lobstein* observes, be seen to present this condition in the vicinity of chronic ulcers or of old cicatrices. As the inflammation advances, the connection between the membrane and the bone becomes more lax, and the effusion may exhibit a purulent character; or the separation may have been so sudden and extensive, especially in adynamic individuals, that, as Dr. Copland believes, before suppuration has time to supervene, gangrene of the periosteum and necrosis of the bone result.

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* "Anatomie Pathologique," vol. ii. p. 83.

In chronic periostitis there is a much less vivid injection, but the swelling is more considerable; the membrane appears dense, opaque, or granular, and is generally adherent to the bone.

The outcome of simple periostitis is either suppuration or ossification.

Periosteal Suppuration.—The periosteum may exhibit considerable swelling and a high degree of inflammation before suppuration takes place; when it occurs, it is sometimes very rapid. Pus accumulates between the periosteum and the bone and in the layers of the membrane. In the former case superficial or total necrosis of the bone results. If a part only is affected, an area of superficially dead bone is found, limited on all sides by thickened and inflamed periosteum. If the suppuration be very diffuse, the whole shaft of a long bone, for instance, may be separated from the periosteum. The resulting necrosis is not to be regarded as wholly caused by cutting off the blood supply of the bone; hence there is nearly always simultaneous inflammation of the subjacent bone tissue itself, or ostitis. It is probably not strictly correct to speak of the separation of periosteum from bone, except by violence, since, in cases of apparent separation by inflammation, the superficial layer of bone is removed with the periosteum.

Periosteal Ossification.—It must be remembered that the formation of bone is, so to speak, a normal function of the periosteum, and this function is called into play either in the course of normal development, or to replace lost bone in normal conditions of the human body. In the lower animals, it has been shown by Ollier, to persist even in pieces of periosteum entirely detached from their original site and grafted in other parts of the body. The property seems to reside chiefly in the layer of formative cells (medullary cells of some authors) forming the deepest layer of periosteum, or sub-periosteal layer; but bone may be formed in the outer layers, or even in connective tissue. The reproduction of bone by the periosteum is always accelerated by hyperæmia, and the state of intense activity which constitutes inflammation. Hence the ossification is commonly regarded as a result of inflammation. Inflammation of the periosteum leading to the formation of bone, may be distinguished as ossifying periostitis. It is seen in the repair of bone after injury, and especially after fracture, and also accompanying necrosis. Chronic periostitis tends to enlargement of the bone, unless necrosis of the latter intervene; and, even then, the ultimate result is usually thickening. Numerous specimens exhibiting the process are to be found in our pathological museums. Moreover, a large number of the osteophytes and other osseous growths on bones are doubtless connected with periostitis. A remarkable fact connected with periosteal thickening is the occasional formation of bone in the connective tissue near bone, though unconnected with it. These detached bony masses are called *parosteal* by Virchow. They may be compared with the islands of bone sometimes found in the tumours of the jaw called epulis (p. 536).

Specific Forms of Periostitis.—Syphilis produces certain special forms of periosteal inflammation; others are attributed to rheumatism; and another has, from its indolent and degenerative character, received the name of scrofulous.

The rheumatic and syphilitic forms chiefly affect the more superficial parts; the periosteum of the skull, the sternum, and the tibia being the points most commonly attacked. Syphilitic inflammation, or at least that which occurs in the course of syphilis, whether as a result of the virus or of the mercurial treatment, is apt to occur in numerous detached spots, at which tumefaction, induration, the formation of new osseous matter, sometimes leading to considerable thickening, and necrosis present themselves. The periosteal gummata will be spoken of in detail further on. Rheumatic periostitis is more likely to occur in the vicinity of the joints, and is acute in its character. Chronic, so-called rheumatic, affections have been described in speaking of joints.

The strumous or scrofulous form of periostitis is that frequently met with in cachectic and scrofulous subjects; it is of a sluggish character, causing greater thickening of the membrane and closer adhesion to the bone, followed by suppuration in the tissue, and underneath or upon it. Scrofulous periostitis is sometimes followed by exfoliations of the subjacent bone; in these cases, there appears to be generally a coincident formation of new bone, sufficient to prevent not only a loss of strength, but even a deformity. One of the forms of so-called scrofulous periostitis not unfrequently met with is that giving rise to the severer forms of panaritium or whitlow, the periosteum of the phalanges being the seat of inflammatory action.

OSTEOMYELITIS AND OSTITIS.

Osteomyelitis.—By this term is meant inflammation of the red osseous medulla and of the pulp contained in the cancelli of spongy bone, since these two are essentially the same structure. The constitution of the red pulp has only lately been carefully investigated. It is found to resemble in some respects the splenic pulp, being composed of lymphoid corpuscles and vascular tissue, which is capable of great and variable distension. It also contains large compound, or myeloid cells. It is probable that this is also one of the seats of formation of white blood corpuscles. By absorption of fat it passes into the yellow medulla.

With reference to its pathological appearance, it is important to remember that simple redness or vascularity does not always indicate inflammation, since this is seen in some bones in atrophic conditions of the body, when the corpuscles waste and the vessels expand to fill their place. A creamy, or mottled condition, is seen, on the other hand, in conditions of abundant nutrition when the lymph corpuscles are very plentiful, as in the spleen in similar conditions. The signs of inflammation of the medulla are a vivid or scarlet injection, with purulent infiltration,* small spots of suppuration, or scattered abscesses. If the inflammation be intense the whole spongy tissue may be softened and break down at a touch, leaving a thin external shell of bone only. Part or whole of the fat is at the same time absorbed, so that we find red medulla at parts where yellow normally occurs. If localized this process may of course lead to abscess, identical with that described under the head of ostitis. This disease is of great importance, as it frequently leads to pyæmia, a fact easily understood, if we remember the intimate relations of the medullary tissue to the blood; and this, whether we suppose a specific poison to be present or not. This connection has been especially noticed in military surgery.†

Ostitis.—The first stage of this form of bone inflammation has already been described. In its second stage it either passes on to hardening or sclerosis, or else to suppuration. The sclerotic ostitis consists of the formation of fresh layers of bone around the vessels. It must be regarded as essentially the same process as goes on in the ossification of vascular tissue without formation of cartilage. In bone thus affected the Haversian canals are found small, and the texture particularly hard and ivory-like. The spongy part of the bone may also be condensed by a similar process, new osseous matter being deposited on the inner surface of the cancelli; and may thus come to be quite as hard as the compact part of the bone. The medullary cavity may be partly filled up in the same way. This process is seen in the ends of long bones in chronic rheumatic arthritis, formerly described, also as an accompaniment of other forms of inflammation of bone, and in some cases of repair, such as the closing of the medullary canal after amputation. Suppurative ostitis, or abscess, is developed out of the rarefactive stage of inflammation by filling of the dilated spaces and canals with pus. The partitions are gradually softened and break down, till a cavity is formed just as in abscess of soft parts. There may be very general suppuration, so that a considerable portion of the bone is destroyed; in which case it is always complicated with osteomyelitis; or else limited suppuration, forming an abscess cavity. The latter affection was first clearly distinguished by Sir Benjamin Brodie. In the first case recorded by him the lower end of the tibia is described as vascular on the surface and hardened around the cavity, which was smooth, and filled with dark coloured pus. There is a distinct pyogenic membrane. These abscesses are small, and situated near the ends of the long bones. If the pus in them should become inspissated and caseous, it would obviously become what is called a "scrofulous deposit in bone."

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* See figure in "Trans. Path. Soc.," vol. xvi. pl. 12.

† Holmes, "St. George's Hospital Reports," vol. i.; but see "Reports of the Army Medical Department," vol. xiii. p. 244.

The distinction between ostitis and osteomyelitis is by no means clear. In ostitis the elements concerned are not the bone-cells, but the embryonic cells derived from the medulla, and these collected in large masses produce the changes of osteomyelitis, so that ostitis is myelitis of compact bone. On the other hand, though we might speak of sclerotic or productive osteomyelitis forming bone in the medullary cavity, that process is more often spoken of as ostitis. The embryonic cells may, in fact, in either case, degenerate into pus corpuscles, producing abscess, or they may take the character of osteoblasts, and new bone be formed.

NECROSIS.

By necrosis is meant the death of a portion of bone, just as in necrosis or gangrene of soft parts. The death of a portion of bone is caused by some influence interfering with the supply of blood to it. The circulation may be interrupted by violent lesions, as fracture or crushing (by separation of the periosteum, it is supposed), by thrombosis or other obstruction of the nutrient artery, or, as Förster says, by excessive cold or heat. This is known as primary necrosis. It may also be, and is much more frequently a consequence of inflammation of bone in either of its three forms, ostitis, osteomyelitis, or periostitis.

Special forms of necrosis are due to the poison of phosphorus, or to mercury in certain constitutions. These, as well as the scrofulous and syphilitic forms, appear to be really consequences of inflammation.

Necrosis attacks chiefly the compact tissue, and is, therefore, most frequently met with in the shafts of long bones, from which it rarely extends into the epiphyses, though instances of its doing so are on record, as in the case of the tibia; in the instance from which Fig. 190 is taken, the process is seen to extend through the neck of the femur into the head of the bone, and even to affect the innominatum. The relative frequency of its occurrence in different bones is stated to be in the following order:—The tibia, the femur, the humerus, the cranial bones, the lower jaw, the last phalanx of the finger, the clavicle, ulna, radius, fibula, scapula, upper jaw, pelvic bones, sternum, and ribs.

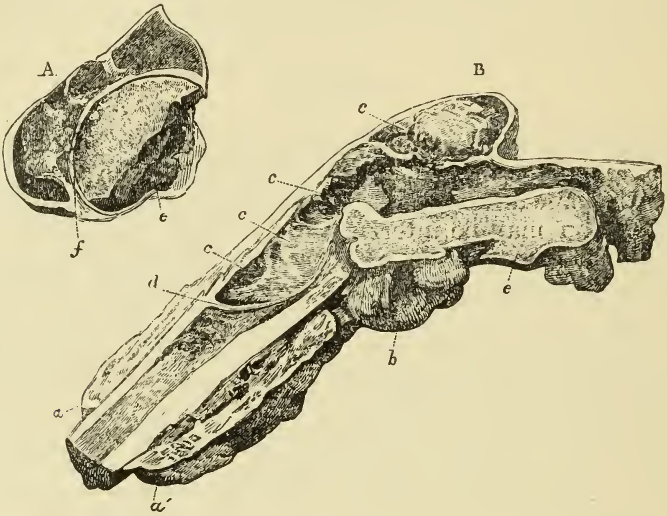
Dead bone is known by being white, opaque, dry, and sonorous when struck; also by being separated wholly or partially from its connection with surrounding parts. When exposed to the air, even indirectly, it often becomes black or discoloured. It often shows traces of past inflammation; being either condensed (sclerotic) or rarefied.

Necrosis may be superficial or central, total or partial.

The necrotic portion is not at first separated from the healthy or less altered bone; but a process is set up which ends in its complete demarcation, like the inflammation which cuts off a gangrenous external part of the body.

In the process of separation the following changes occur:—All round the necrosed portion, that is to say, at its margins and at the part where its surface is exposed to that of the healthy bone, the latter undergoes a gradual expansion, or rarefaction of its tissue, by the enlargement of its Haversian canals; assumes a rosy colour, and becomes succulent. It gradually acquires an areolar structure, and is thus more rarefied; at length it disappears altogether, and a red, soft, spongy substance, a layer of granulations, occupies its place. This change is, in fact, inflammation of bone, which gives rise to suppuration and granulation. The bony tissue, beginning with the Haversian canals, is dissolved by the matter secreted within them, while the granulations which continue to grow fill up the spaces thus left. The immediate result of this process is the formation of a furrow of demarcation, which encircles the margin of the dead bone, and is filled up with granulations and pus, till at length the sequestrum is separated from the surrounding bone by a suppurating groove, which always communicates

Fig. 190.



Necrosis of the head of the femur, acetabulum and shaft, subsequent to amputation.

A. Necrosed acetabulum and head; completely ankylosed and broken off at *e* from the neck. *f*. Remains of the articular surfaces closely united. B. Neck and upper part of the shaft. *a*. New bone. *b*. New bone undergoing necrosis. *c*. Bone still containing blood-vessels, and in various stages of inflammation. *d*. A membranous septum, marking the boundary of the dead and living bone.

with the surface by one or more openings, termed *cloacæ*. It is very rare for dead bone to be enclosed on all sides by living bone; generally it is in contact with the periosteum on one side. The changes which this membrane undergoes accordingly become an important factor in the separation of the dead bone.

One of two events may happen; either the periosteum dies with the bone, in which case the dead surface of bone is exposed, or it becomes thickened and undergoes ossification. In the former case the limiting inflammation of bone is all that is necessary for complete separation, and the dead bone exfoliates. In the latter case, not only is the dead bone cut off on the periosteal side, but a process of repair sets in by which the loss of bone is replaced. The process is best seen when the whole or large part of a long bone suffers necrosis, specimens of which are to be found in all museums.

Simultaneously with the formation of the furrow of demarcation between living and dead bone, a similar space, filled with pus and granulations, is formed on the periosteal side, so that the periosteum is apparently detached, and the sequestrum isolated on all sides. Billroth contends that the periosteum is not, strictly speaking, detached from the bone, but that what occupies its place is partly the superficial subperiosteal layer of bone, partly the external layers of periosteum, while the intermediate or truly fibrous periosteum simply perishes. Goodsir always held that some bone was detached with periosteum. However this may be, there is a thick structure in the place of periosteum, which, as it becomes separated from the bone, at the same time undergoes ossification; this process taking place, as Billroth states, also in the adjacent connective tissue. In consequence, long before the dead bone is separated, there is a large amount of periosteal bony growth, and ultimately, when the isolation of the sequestrum is complete, it is surrounded on all sides by a bony case, but separated from it by an intervening space, in which are pus and granulations, and which communicates with the external surface by cloacæ or sinuses. The new bone thus produced is at first spongy or callus-like, but becomes denser, and ultimately strong enough to replace the perished shaft.

This condition often necessitates surgical operation. If the sequestrum be removed by art, the cavity in which it lay becomes filled up by endosteal ossification; and what is practically a new shaft, always larger than the original, and most probably always solid, without any medullary cavity, is left. In other bones the details of the process will of course be different. A remarkable instance of the capabilities of an unaided constitution occurred to us while investigating the effects of the powers of phosphorus in producing necrosis of the maxilla. A man came under our notice, in whom the destructive process had caused death of the body and rami of the lower jaw to such an extent that the entire bone, with the exception of the condyloid processes, came to lie loose in the cavity of the mouth; a new jaw having, in the interval, formed underneath, and not, as is usually the case, as a capsule to the necrosed part. The patient's mouth not being of sufficient size for the extraction of the bone, he sawed it across with his own hand, and then extracted it with ease.

By the erosive action of the surrounding granulations, the mass of dead bone is to some extent diminished, and its surface becomes rough; but this alone is never sufficient to remove a sequestrum completely. In fact, the removal of absolutely dead bone has been denied by some pathologists, who believe that the space around the sequestrum is formed entirely at the expense of the surrounding bone.

This point will be discussed under "caries." The controversy is a very old one.

Central necrosis means necrosis commencing in the inner part and spreading outwards, layer by layer. In this case dead bone may be enclosed in a shell of the original living bone; but this form is rare, and generally the external shell is of new formation, as described.

CARIES.

The name caries is used to mean substantially the same as ulceration, that is, perishing of tissue and removal of the products; but though we generally speak of ulceration only on a surface, caries of bone may be, though rarely, central as well as superficial. Some confusion has arisen from applying the term specially to scrofulous and syphilitic processes, which we shall speak of separately.

Superficial ulceration (says Mr. Holmes) is distinguished by the following characters:—The periosteum is loosened from the surface, and, if the disease is advanced, will be found much thickened, and converted into a villous mass of a pink colour, resembling a layer of granulations. This substance adheres very loosely to the surface of the bone, and, when lifted up from it, is found to fit into depressions, which seem to have been hollowed out of the bone by the agency of the granulations. The bone at a very slight depth underneath is found, in most cases of healthy inflammation, of the ordinary consistence of cancellous tissue, which it resembles in structure even in those parts which ought to be compact. The ulcerated surface is superficially excavated, much softened, and easily broken down. The interior of carious bone is softened by inflammation, its cancelli enlarged, and filled with the products of softening and disintegration. These products are principally oil globules, blood, and other *débris* of the soft tissues, and granular inorganic materials having the same chemical composition as the salts of bone. Caries is generally accompanied by more or less plastic or organizable effusion in the bone in the neighbourhood of the ulcerated spot, leading to condensation of the deeper parts of the bone, and to periosteal deposit of bone on the surface. This is only seen in healthy or sthenic inflammations.

Central caries in spongy bone begins with hyperæmia of the medulla, wasting of its adipose tissue, and production of pus corpuscles, but not in very great number, till a brownish red pulp is formed, and by absorption of the trabeculæ, an excavation; which is distinguished from an abscess by not containing true pus. The process may then extend outwards, layer by layer, till the whole bone is destroyed (Förster). Granulations are sometimes formed which assist the absorption, or it may pass into actual suppuration.

Necrosis may accompany either form of caries; if on the surface, small fragments of dead bone are shed off; if in the substance, sometimes a larger necrotic portion is found in the middle of the carious mass. The surface, if exposed to the air, is often blackened.

Caries affects particularly the cancellous bones, as the vertebræ, the epiphyses of long bones, the bones of the hands and feet; but the cranial bones are sometimes affected. It is almost always a chronic process, but some very rapid, almost gangrenous cases, have been called phagedænic caries.

The effects of caries on the bones are very obvious in macerated specimens. The external shell is either destroyed, excavated, or perforated, the cancellated tissue rarefied, its partitions attenuated, and larger or smaller cavities formed, so that the whole has a ragged appearance. Sometimes, in consequence of simultaneous enlargement, the appearance is very much that of osteoporosis. Outgrowths of bone are sometimes seen around the carious patches.

Minute Changes in Caries.—In all cases of absorption of bone, the surface on which absorption is taking place will be found hollowed out in hemispherical or curved depressions—the lacunæ first described by Howship. More minute excavations, seen under the microscope, give a concave sinuous outline to the edge of a section. These hollows are filled during life with granulation-tissue, and to this the most recent observers assign the chief part in absorption of the bone. This view is supported by the fact that, not only dead bone, but plugs of ivory driven into living bone, may be partially excavated and absorbed, and that on the surface of these plugs similar excavations are seen. How granulations absorb bone, we do not know.

Virchow rejects this explanation, though admitting the excavation of ivory plugs by some such means. He believes that caries consists in a breaking up of the bone into its cell-territories, *i.e.*, portions depending on each bone cell, and softening of these territories individually, while the cells, both of the bone and the medulla, proliferate and take on a new development; the intercellular substance being cast off in fragments. The lacunæ are formed by softening of successive cell-territories. The process is essentially (according to him) a degenerative ostitis, by which the osseous substance becomes converted into a soft uncalcified tissue, which may, according to circumstances, be either a degenerating mass with fatty elements, or a richly cellular structure with newly-formed elements. The latter may, like the medulla, become luxuriant in its growth, and proliferate on the surface in the shape of a granulation. The granulations, then, are to be regarded as originating in bony tissue.*

Billroth, on the other hand, believes that the bone-corpuscles are simply passive till set free by the softening of the surrounding substance, when they may possibly proliferate; but he regards the solvent action of granulations as a fact, though an unexplained one.

It is probable that the process will be better understood when it is brought into connection with the physiological absorption of bone in the formation of medullary cavities or other hollows, and in the changes of form which bones undergo. These have been lately studied by Kölliker,† who finds that in each of the Howship's lacunæ (which occur in normal absorption also) is a large compound cell, a myeloid or "giant cell," to which he gives the name of *osteoclast*. These osteoclasts he believes to be the essential agents in the process of absorption, with which the fixed bone cells have nothing to do; and this is shown by the fact that dead ivory pegs (as in the experiments of Langenbeck) become excavated with lacunæ, containing osteoclasts. Similar cells have been found in provisional callus which undergoes absorption, and in bone which is wasted or absorbed through pressure (either from tumours, or produced artificially); but at present little has been done to connect these bodies with the process of caries and necrosis.

Scrofulous Ostitis, or Caries.—The disease of bone, called scrofulous, though often taken as the type of caries, is in reality not quite so simple, and would be better called scrofulous ostitis. It has been regarded as tubercular. The affection, which we call scrofulous in the sense defined above (p. 91), is especially seen in the cancellous bone, and at the extremities of the shafts of long bones, whence it not unfrequently extends to the joints, producing the scrofulous or fungous arthritis already referred to. In early stages of scrofulous caries, the bone (says Mr. Holmes) "is soft, light, and oily . . . . the cancelli are large, and charged with a red jelly-like mass." M. Ranvier regards this as a preinflammatory stage, and dependent on the destructive fatty change in the bone cells. The inflammatory changes he regards as subsequent to these. But when once established the disease is essentially a degenerative inflammation, ending either in the production of a cavity containing curdy pus, which has the character called scrofulous, with few formed elements, and much molecular *débris;* or else by a probably slower process in the caseous infiltration of portions of cancellous tissue; or else in the necrosis of detached masses of bone, which become enclosed in a cavity. If the cavity open into a joint, something not unlike a pulmonary vomica is produced. Florid granulations often sprout from the edges, and condensation of the surrounding tissue is often seen. Sometimes new bone is produced from the periosteum; as we have repeatedly seen in so-called scrofulous caries of the vertebræ. Calcareous infiltration of the pus or cheesy matter may take place, forming chalky masses, easily distinguished from bone. Suppuration of the periosteum is very often induced, and chronic abscesses formed, of which a familiar instance is the well-known psoas abscess, extending from caries of the vertebræ. Such abscesses, wholly or almost healed, are sometimes found after death, containing cheesy or putty-like matter, formerly regarded as necessarily tubercular.

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* "Cellular Pathologie," p. 523, 4th edition, 1871.

† "Die normale Resorption des Knochengewebes," Leipzig, 1873. "Quar. Jour. Mic. Science," xiii. 89; xiv. 87, etc.

Connection of Caries with Tubercle.—Some pathologists, as for instance Nélaton, have spoken of this disease unconditionally as tubercular. Grey miliary tubercle is found only in the very rarest instances. Yellow matter, formerly called crude tubercle, is found, but by no means constantly, and of course is not necessarily tubercular. Tubercles are not found in the neighbourhood; so that the positive connection with tubercle is slight. On the other hand, the disease is not unfrequently associated with tubercular disease of other parts. Billroth found tubercular or cheesy products in other parts of the body, occurring in 54 per cent. of his cases of caries. Mr. Haward's statistics ("St. George's Hospital Reports," vol. iv.) give a very different result; out of 134 cases of chronic bone and joint-disease in children usually called scrofulous, he found only seventeen with other signs of scrofulous disease, and only nine with signs of actual tubercle; while, out of eighty-five cases of children admitted in the hospital for various tubercular diseases only one had any bone disease. Mr. Holmes thinks the masses called crude tubercle in bone are of inflammatory origin, and that rare as tubercle in bone is generally supposed to be, it is in reality still rarer. The question is evidently the same as that respecting the origin of cheesy masses in the lung, the lymphatic glands, the kidney, and other parts.

HYPERTROPHY AND ATROPHY.

It is doubtful whether true simple hypertrophy occurs, but enlargement of bones in all their dimensions is seen in rare cases as a sort of monstrous growth, without any obvious cause. Inequality of the sides of the body, or of the parts of the hand or finger, results. Of course with this must be associated corresponding enlargement of the soft parts. Inflammation, both of the periosteum and of the substance causes overgrowth or *hyperostosis*, by which the bone is thickened without becoming necessarily longer or wider. The surface is usually irregular, sometimes still further complicated with bony growths, or osteophytes; and from interstitial sclerosis, the bone is also often heavier. This is seen chiefly in the long bones of the limbs and in the cranial bones. The process appears to resemble normal periosteal growth, consisting in the ossification of successive layers. Sometimes a process, which appears similar, occurs without any inflammation, and under this head must be brought certain extraordinary cases of bony development, where thickening of the whole bone, without special growth on the surface, is observed.* Dr. Wilks records a case where the skull became $\frac{1}{2}$ to $\frac{3}{4}$ inch thick; the femur measured $8\frac{1}{2}$ inches in circumference above the condyles, and weighed 2 lbs. 14 ozs.

The process has a certain resemblance to osteoporosis, into which it is possible that Dr. Wilks's case might have developed.

A remarkable lobulated or botryoidal form of hyperostosis sometimes attains a great size. It is composed of compact, though not necessarily ivory-like bone. It is chiefly seen in the cranial bones. A remarkable instance, producing frightful deformity of the face, is described and figured in the "Pathological Transactions" (vol. xvii. p. 245). It affected, beside the cranium, the hyoid bone and the fibula. On the latter was formed a solid tumour, more than 5 inches in diameter. The monstrous deformity produced in such cases has led Virchow to propose for them the name *Leontiasis ossea*. Other celebrated cases are on record.†

Simple elongation of the long bones appears to be the consequence of some inflammation occurring before growth was complete; the permanent result of a temporary affection. In the leg this usually depends on elongation of the femur. Sometimes the tibia, without the fibula, is lengthened, and then necessarily becomes curved.

Physiological hypertrophy is seen in cases of great muscular development, where the bones generally, and especially the ridges for attachment of muscles, are found enlarged.

Hypertrophy of the skull-cap is frequently, though by no means invariably, a concomitant of wasting of the brain. It is also said to occur in persons who go bareheaded, from hyperæmia of the scalp. Condensation of bone, from interstitial growth, must be considered a kind of hypertrophy. It usually results from some kind of inflammation. This condition is seen in the "eburnation" of the articular ends of the bones, in chronic rheumatoid arthritis. We have another instance in the minute structure of gouty bone, where, as we are informed by Mr. Ure,‡ the Haversian canals are enlarged, and choked up with cretaceous matter, which also lines the medullary canal; the osseous corpuscles are also found to be larger than usual, rather irregularly scattered, and less distinct, and their canaliculi loaded with chalk, which is shown by analysis to be true carbonate of lime, and not like the tophi of adjoining articulations, urate of soda.

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* "Trans. Path. Soc.," vol. xx. p. 273.

† Virchow, "Krankh. Geschw.," vol. ii. p. 21.

‡ "Lancet," 1847.

Atrophy.—Simple atrophy is less rare than simple hypertrophy; but this condition not unfrequently results from several causes. "Inflammation, fatty degeneration, disuse, or injury," are mentioned by Mr. Holmes. It is also distinguished as *concentric atrophy*, in which all parts of the bone waste equally, the medullary cavity becoming smaller at the same time as the walls become thinner; and *excentric atrophy*, in which the cavity enlarges at the expense of the external portions. The latter condition may be combined with or merge in that of osteoporosis.

The condition known as concentric atrophy results in limbs from disuse, whether in consequence of dislocation, stiffness of the joints, ankylosis, or paralysis. Partial atrophy may be the result of fracture or other injury to the bone, as has been thought (though Mr. Holmes doubts this), from injury to the medullary artery.

Excentric atrophy does not necessarily cause any diminution of the external dimensions, but the bone is rarefied; both the cancelli and the medullary cavity being enlarged, and the partitions absorbed, while the external wall becomes reduced to a mere shell. In this condition the bone is extremely light and brittle; the latter state has given rise to the name *fragilitas ossium*, and may give occasion to so-called spontaneous fractures. In flat bones all the cancellous tissue may be absorbed, and the tables come in contact.

The causes of excentric atrophy are senile decay and various cachexiæ. It is usually accompanied by fatty degeneration, and the bones, when removed and macerated, are yellow and oily. This property is noticed by museum collectors in the bones of animals from menageries; which show also the results of muscular inactivity in the smooth surface, and wasting of the muscular ridges.

Two peculiar conditions allied to atrophy must be spoken of separately—viz., osteoporosis and mollities ossium.

OSTEOPOROSIS.

In the rarefied state of bone to which the term of osteoporosis was given by Lobstein, the affected bone presents an increase of size, and a diminution of density, owing to the tissue being expanded; the surface of the bone is irregular and very porous. When the bone is fresh, signs of hyperæmia and inflammation are present, the medulla being vascular and rich in cells, the periosteum swollen and injected; but on macerating the bone its actual substance is found diminished, the gradual expansion inducing a thinning of the surrounding osseous layers, and eventually a communication between adjoining cavities. The condition may affect the compact or cancellous tissues alone or together. When the cortical layers are the seat of the change, the appearances may induce the resemblance of caries. Lobstein describes the surface in this case as being covered with a multitude of longitudinal fibres, resembling those of a fœtal skull. He attributes them to the development and the action of the periosteal vessels, which hollow out for themselves channels in the osseous substance. When this variety of cortical rarefaction is raised above the surrounding tissue, it resembles one form of osteophyte, and many of the preparations of caries preserved in museums are referable to the same head. The rarefaction of the osseous tissue, which constitutes osteoporosis, though often inducing a considerable increase of bulk, is essentially distinct from the process giving rise to the formation of exostosis, in which the generation of new osseous matter is the characteristic feature; the former being essentially an atrophic, the latter an hypertrophic condition. The term spina ventosa, though one which has nothing but the prestige of antiquity to recommend it, is applied, among others, to affections belonging to the class just considered. We will not seek to perpetuate it by defining its characters, as it is an arbitrary designation, without any acknowledged and established meaning. There appear to be two chief forms of this disease. (*a*) That in which the distinction of medullary cavity and compact substance is lost, all being converted into a spongy or cancellated structure. This may affect part or whole of the bone. (*b*) In another form the medullary cavity remains, but the shell is converted into a thick mass of spongy substance. Enormous spongy expansion of particular parts is sometimes produced by cancers or other tumours, which will be spoken of again.

MOLLITIES OSSIUM.

Mollities ossium, osteomalacia, or malacosteon, is a disease regarding which the views of authors differ; though it is no doubt essentially distinct from other known osseous maladies. It is a very rare complaint.

The disease consists, as the term indicates, essentially in a softening of the bones, brought about by an absorption of the earthy matter, and the substitution of a large quantity of fat or gelatinous material. It is an entire perversion of the process of nutrition, in as far as regards the skeleton, the earthy phosphates being eliminated from the system by the kidneys, and necessarily induces great pliability and fragility of the bones. Lactic acid has recently been detected both in the bones themselves, and in the urine of persons affected with this disease. The bones are so much weakened that so-called spontaneous fractures often occur.

Kilian* treats of mollities ossium as presenting two varieties—the waxy (cerea), and the fragile (fracturosa). In the former, the bones generally, but especially those of the pelvis, present a dirty dark-yellow colour. They lose their transparency in the middle, while their weight is not much diminished, and they become flexible, like wax; in the second, the bones present a snowy whiteness, and a light, transparent, open texture, rendering the bones so fragile that they give way under the mere pressure of the finger. The first kind of bones do not dry clean, but remain greasy, the second dry quickly, and give no greasy feel. Both varieties, according to Kilian, exert the same influence upon the pelvis in regard to the distortions that are produced. These he describes as consisting in angular deflections of the individual bones, and a mutual approximation of the bones in the conjugate and transverse diameters. If Professor Kilian's view regarding the two species of the malady be correct, it is probable that they would be distinguished by their chemical constitution, and this may be assumed as the reason why the chemical analyses of the bones affected with osteomalacia have yielded results so widely apart. Thus, in one of the two remarkable cases detailed by Mr. Solly,* the analysis of the affected bone by Dr. Leeson showed 100 parts to contain—

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* "Beitrag zu einer genaueren Kenntniss der allgemeinen Knochenerweichung," &c., Bonn, 1829; and "Die Geburtslehre," &c., vol. ii. p. 367, 1840.

Animal matter	18·75
Phosphate and carbonate of lime . .	29·17
Water	52·08
	100·00

The chemical examination of a case (detailed by Dr. Ramsbotham, in the "Reports of the Pathological Society")† by Dr. Garrod, yielded—

Fatty matter	20·35
Gelatine yielding matter	58·37
Carbonate and phosphate of lime, and phosphate of magnesia . . .	21·28
	100·00

In the former analysis, we see nearly eight parts more earthy matter than in the latter; nor can it be objected that the analyses were made at different stages of the disease, because, in both instances, the patients from whom the specimens were taken had succumbed to the malady. The analysis given by Dr. Bostock reduces the amount of earthy matter to a yet lower figure. In a specimen that he examined, he found the proportion to be, in 100 parts of bone—

Jelly and oil	22·5
Cartilage	57·25
Earthy matter	20·25

The microscopical appearance of the bone, according to Rindfleisch, is as follows:—The trabeculæ (of the cancellous portion) show only a central axis which still retains the perfect bone structure. Outside this is a zone of fibroid material, with hardly visible bone corpuscles. having precisely the appearance of bone decalcified by hydrochloric acid; and it is plain that a process of decalcification spreads from the medullary and cancellous cavities into the solid bone. The tissue thus decalcified is partly absorbed, partly softens into a mucous or colloid material, which with fat occupies the place of the removed bone tissue. The medullary tissue is described by Rindfleisch as extremely vascular, with dilated vessels, and showing occasional hæmorrhage, but with no proliferation or special change in the medullary cells; the general appearance being rather that of passive than of active congestion. From the Haversian canals the process spreads outwards, and from the cancelli and from the medullary cavity also outwards, till only a thin shell of bone is left, which perhaps escapes because nourished by the periosteum. It seems, then, that the process is clearly one connected with the medulla, though precisely in what way is not known.

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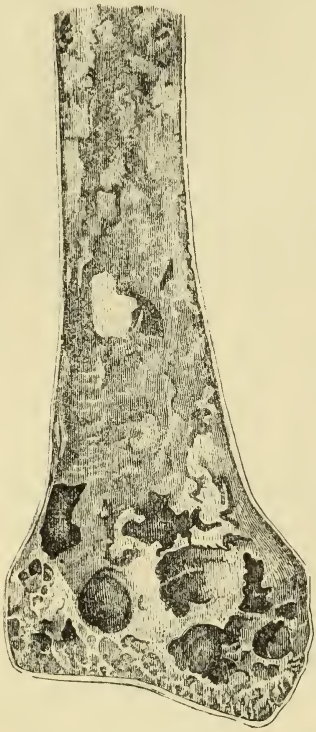
* "Med.-Chir. Trans.," vol. xxvii. p. 435.

† "Reports," &c., vol. xxv. 1847-48.

FIG. 191.



Section of a femur from a lady, aged thirty, affected for some years with mollities ossium. The walls of the bone are thin, soft, and flexible; the place of its medullary and cancellous tissue is occupied by soft jelly-like transparent matter of various shades, of yellow and pink, some of it deep crimson; a similar kind of fat appeared to be diffused through the proper texture of the walls.

(St. Bartholomew's Museum, i. 233.)

As the bones of the trunk are especially liable to be attacked, the individual affected becomes reduced in size from the collapse of the vertebral column. A tall subject may thus be converted into a dwarf; an instance of which we had an occasion of seeing in the Clinique of Professor Kilian, of Bonn. The individual was a married woman whose stature had diminished in this manner, and was doubly interesting from having in this condition become the subject of a successful Cæsarean operation. It was subsequent to her recovery from this ordeal that we saw her, and that she was supposed to be again pregnant. Dr. Greenhalgh enabled us to examine a similar instance, in which the disease was also followed by contraction of the pelvis, rendering the Cæsarean section necessary. Adults, and especially females, are the subjects of the malady. It attacks women chiefly after they have commenced child-bearing. Mr. Curling* has collected sixteen cases, thirteen of which occurred in females, and three in males. Eleven were fatal between thirty and forty. In none it showed itself before puberty; but two patients were above fifty years of age. Several of them were delivered of children during the progress of the complaint. It is not associated with any particular lesion of the viscera; neither can an hereditary or idiopathic taint, or diathesis, be traced, by which the peculiar symptoms could be explained or referred to a known type.

It seems probable that certain cases of so-called mollities ossium are essentially primary cancer of the skeleton. We have ourselves seen a case of this kind.

In a well-marked instance of mollities ossium, of which the history was given to the Medico-Chirurgical Society by Dr. T. K. Chambers,† and which occurred in a female, aged twenty-six, the bones throughout the system were soft and yielding, so that a sharp knife could readily pass through them.

A peculiar cystic condition of bone is thought by Rindfleisch to be possibly a sequel of mollities ossium. The formation of cystic cavities is stated to represent a stage in the development of that disease at which arrest of the morbid process takes place. This cystic degeneration affects those parts of the skeleton, and only those, previously affected by mollities ossium.

RHACHITIS, OR RICKETS.

Rickets differs from scrofulous affections in this respect, that while frequently associated with inflammatory conditions, it is not essentially an inflammation; one of its symptoms is a diminution of the consistency of the osseous texture, but it does not exclusively consist in rarefaction of the bone; nor does it manifest those organic and chemical changes which constitute the peculiar disease known as mollities ossium. Owing to one feature having been regarded by different observers as characteristic of the disease, analogies have been repeatedly set down as proofs of identity. While a general debility is the chief constitutional feature of the disease, a deficiency in the earthy matter of the bones is one of the chief local phenomena, yet the ricketty bone is not simply a soft bone; but it undergoes, during the development and subsidence of the disease, a series of curious and somewhat complex changes. The affection is especially one of early childhood; the first symptoms may be seen as early as four months old, but are not usually conspicuous till towards the end of the first, or in the second year. Guérin found, that of 346 cases, 209 were affected between the first and third years; only three were congenital;

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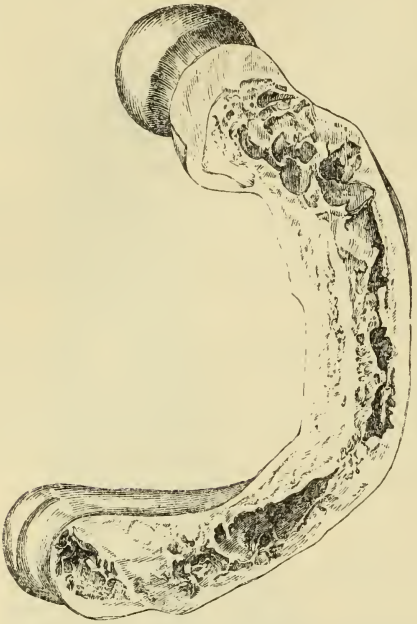
* "Med.-Chir. Trans.," vol. xx. p. 336.

† "Medical Times and Gazette," March 25th, 1851.

and thirty-four occurred between the ages of four and twelve; 148 were males, and 198 females. The alleged congenital occurrence of rickets requires confirmation.

Before any distinct changes in the skeleton take place, other symptoms of cachexia are seen, admirably described by Sir W. Jenner,* in his classical Lectures on Rickets, but which do not concern us here.

FIG. 192.



Section cut with a knife of the femur of a rickety child. The shaft consists throughout of cartilaginous and gelatinous substances, intermixed and disposed in cells; it is observable that a greater quantity of cartilage exists in the middle of the shaft and towards the interior curve than at any other part.

(St. Bartholomew's Museum, i. 34.)

The alterations of bony structures are chiefly the following:—(1) Enlargement of the ends of the long bones, that is, of the end of the diaphysis next to the cartilage, in the bones of the limbs; of the part next to the costal cartilages in the ribs. (2) General softening of all the bones. (3) Thickening of the flat bones, *i.e.*, the bones of the skull, the scapulæ, and the pelvic bones. (4) Deformities, produced by mechanical causes acting on the softened bones, such as curvatures of the spine and the long bones, and alterations in the shape of the thorax and pelvis. (5) Arrest of growth, both of the bones and of parts anatomically or physiologically correlated to them.

(1) The enlargement of the ends of the bones may be considerable. In one of Sir W. Jenner's cases the circumference of the wrist was equal to the length of the forearm, each being 4½ inches (in a child 30 inches high; three years and two months old). The parts thus enlarged are normally somewhat prominent in children, in consequence of the process of ossification which is going on; but the rickety enlargement is generally characteristic. The adjacent cartilage is always somewhat enlarged also on the side next the bone.

(2) There is, undoubtedly, some general softening of the bones, due apparently to a deficiency of lime, and by this their curvature is made possible. Virchow, however, alleges that any considerable curvature in the shaft is always due to some fracture. Rickety bones have been found to contain only 21 per cent. of

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* "Medical Times and Gazette," 1860, vol. i.

mineral matter, while the bones of healthy children should contain 37 per cent. (Jenner).

(3) Thickening of the flat bones is due, precisely as in the swelling of the ends of long bones, to hypertrophy of the medullary pulp, and expansion of the cancellous tissue. The substance of the bone seems mainly to consist of diploe (Aitken). In the cranial bones it makes the sutures appear like deep furrows. It has been supposed that this diminishes the capacity of the cranium. The fontanelles are always late in closing. A peculiar form of disease of the cranium has been described by Elsässer,* as occurring in rhachitis, and characterized by softening, thinning, and perforation of the occiput. The bone is atrophied, soft and porous; and numerous openings are observed along the lambdoidal suture, and in the body of the bone, with the exception of the occipital protuberance. The perforations may amount to as many as thirty; and, in place of bone, they are filled up only by the dura mater and pericranium, which are adherent to one another. The affection commonly manifests itself between the third and sixth month of infant life; the child exhibiting much restlessness, and a fear of all contact with the occiput.

(4) The deformities produced by rickets are very important. They may be temporary or permanent. Of the long bones, those of the lower limb are most conspicuously affected. The femur is often curved, usually with its convexity outwards, sometimes the contrary; sometimes the bone curves forward with the convexity in front. The beginning of this is seen sometimes before a child walks, and is attributed to the weight of the legs and feet. It increases if the weight of the body is carried on a limb not yet rigid. The curve of the tibia is also usually convex outwards, an exaggeration of its normal curve. When with these curvatures, there is change of direction at the softened epiphysial junction, the whole limb may be curved, forming bandy legs. Sometimes the opposite condition, knock-knee, is seen. Similar curvatures of the bones of the arm are less easy to explain; they are thought to be partly produced by the child supporting its body on its hands in crawling. The attachment of muscles is also, in some cases, the explanation. Thus Sir W. Jenner points out that the humerus is often bent at an angle just at the insertion of the deltoid, simply from the weight of the forearm and hand, when the limb is raised by that muscle.

Deformity of the thorax is caused by falling in of the ribs along a curved line or furrow, just outside the line of union of rib and cartilage. It is formed by the drawing in of the ribs in inspiration, at the point where they are weakest. It has the effect of carrying the sternum forward, and producing "pigeon breast." The lower ribs are prevented from collapsing by the resistance of the liver and spleen. By these alterations, the capacity of the thorax is greatly diminished, and dyspnœa results.

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* "Der weiche Hinterkopf, ein Beitrag zur Physiologie und Pathologie der ersten Kindheit." Stuttgart, 1843.

The clavicles are very frequently deformed, being bent at an angle in two spots, one near the attachment of the pectoralis major and sternomastoid, the other near the acromio-clavicular articulation. The former is attributed to muscular action in raising the arm, and also to bearing the weight of the body on the arms.

Curvature of the spine is chiefly in the lumbar region, or general. If the latter, it extends from the first dorsal to the last lumbar vertebræ, and is an exaggeration of the natural curve in children that have not begun to walk. Sometimes a concave curve in the lumbar region is associated with convexity (backwards) in the dorsal. This is in children that have begun to walk, and is an exaggeration of the natural double curve of the spine. Angular curvature does not occur; lateral is rare.

The distortions of the pelvis resulting from rhachitis are next in importance to those of the thorax. The irregularities of the pelvic diameters due to this disease are caused by lateral contraction, by an approximation of the acetabula, by antero-posterior narrowing, from an advance of the sacrum, or by an asymmetrical deformity, due to an arrest in the growth of one half of the pelvis. In all these cases the mechanism of parturition will be interfered with in proportion to the amount of malformation, and it is the brim which will be found to be chiefly at fault, though each part individually, or all collectively, may be involved in the deformity. "In most cases of partial deformity at the brim," observes Dr. Ramsbotham,[*] "the lateral diameter is increased in size nearly in the same proportion as the conjugate is diminished: but however much the width from ilium to ilium may exceed the ordinary dimensions, the increased space thus obtained will in no degree make amends for the diminution from the sacrum to the pubes; because it is necessary that there should not exist less than a certain quantity of available room in every direction to permit the child's transit." From the sacrum having to support the entire spinal column, the lower lumbar vertebræ and the base of the sacrum are very apt to be thrown forward where there is deficient cohesion, and the consequence will be a diminution of the conjugate diameter. In this case the diameter of the outlet is frequently found enlarged. The ilia will not present the usual expansion, the crests of the ilia will be nearer to one another than in a normal pelvis, and the female will probably also present a hollow-backed appearance. The sacrum is commonly deprived of its concave form, and exhibits a more rectilinear anterior surface, or as Smellie has observed, the vertebræ that compose it ride over one another, and form a protuberance in the part that ought to be concave. These malformations cause those varieties in the form of the pelvis which have been termed the elliptical, heart, or kidney-shaped, or figure-of-eight pelves. An oblique form in which the ilio-pectineal eminence of one side approaches nearer to the promontory on one side than on the other, was first shown by Nägelé to result from ankylosis of one sacro-iliac symphysis; these pelves present a very characteristic appearance, and look as if one half of the pelvis and the acetabulum had been forcibly pushed over to the opposite ilium; hence the diameter from the sound sacro-iliac union to the opposite acetabulum will be very much diminished, while the interval between the ankylosed symphysis and the other acetabulum will be not only not diminished but even increased. An excellent delineation of this and several other forms of distorted pelvis are to be found in Dr. Ramsbotham's "Atlas of Midwifery." The rickety distortions of the pelvis are probably never met with unaccompanied by spinal curvature; though the latter may occur without materially influencing the pelvic diameters.

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[*] "The Principles and Practice of Obstetric Medicine," p. 39.

(5) The arrest of growth is a very striking phenomenon. Rickety children, though they may quite outlive their complaint and become strong and muscular, never become tall. Their short stature depends not only on curvature, but on actual shortness of the bones from arrest of growth.

MINUTE CHANGES IN RICKETS.

When the swollen end of a bone in rickets is cut into we see the chief morbid appearances at the junction of the bone and the cartilage. Here there is normally in children a transition zone, made up partly of altered cartilage, which is bluish, and partly of red spongy tissue. This zone is immensely enlarged. The changes in the cartilage are found to extend over perhaps twenty or thirty rows of cartilage capsules, instead of one row as usually. The spongy tissue forms a layer several times as broad as it does normally; it contains in its meshes a red pulpy matter, which appears to be the ordinary red medulla, increased in quantity and altered in structure. Moreover, the line of junction, usually uniform, is very irregular; so that at some points cartilage dips deep down into the ossifying zone, while at another calcified portions appear in the middle of the cartilage. The existence of this broad band of flexible uncalcified tissue, at the junction of the epiphysis, is one cause of the distortions which rickety bones undergo.

Examination of the altered zone of cartilage shows that, though so much wider than the normal zone of altered cartilage, the changes in it are essentially the same, the cartilage capsules showing proliferation and being arranged in long rows, as they usually are preparatory to ossification. The spongy tissue, beside its red pulp, is made up of trabeculæ, arranged in the same way as in cancellous bone, but of different structure. They are composed of a tissue formed out of cartilage, which may be called osteoid cartilage, containing rows of proliferating cartilage cells, and angular corpuscles with some resemblance to bone corpuscles, but without canaliculi. The intervening substance is often calcified (as are sometimes the corpuscles themselves), a fact to which Sir W. Jenner attaches much importance. This is calcified cartilage, a very different thing from bone. Kölliker, however, who has been followed by Virchow and Rindfleisch, alleges that there is a direct transformation of this cartilage into bone. This hypothesis is contrary to what the latest researches teach us to believe respecting the ossification of cartilage, and must be regarded as doubtful. The actual process of bone formation, when the rickety changes come to an end, is not known. Some of the trabeculæ appear simply fibrous.

With respect to the red medullary pulp, much of this is normal in structure; but a fibrous transformation of it into connective tissues is described as occurring in some parts, which gives rise to what Virchow calls osteoid tissue, regarded by him as the preliminary stage of bone-formation.

In the periosteum we see the same fact as at the boundary line between bone and cartilage, namely, a great extension of the belt of vascular and embryonic, or granulation-like tissue, which is normally found in periosteal ossification. So vascular and soft is this tissue, that it was formerly imagined to show hæmorrhage between the bone and periosteum. This tissue changes into bone by the usual method of ossification in vascular tissue; but the bone formed remains, according to Rindfleisch, for a long time in a rudimentary condition, like an osteophyte, and is gradually changed into perfect bone. As the thickness of ossifying tissue is here much greater than normal, we see why the bones should be, during the process of ossification, and even permanently, thickened.

These changes may be summed up as showing an abundant formation of the materials which precede ossification, but no bone production. Nor is this simply due to absence of lime, since this is deposited, though not in the right way to form bone.

CHAPTER XLVI.

ADVENTITIOUS GROWTHS IN BONE.

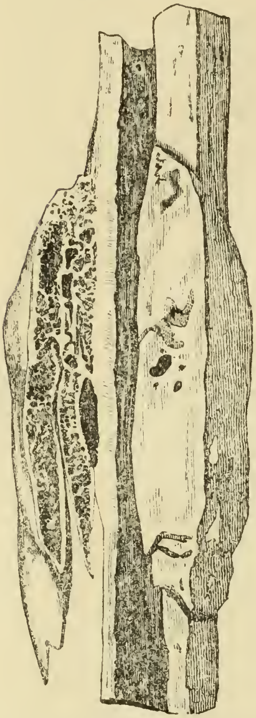
EXOSTOSES.

Osseous growths, consisting of true bone, are divided into exostoses and osteophytes; the difference being marked rather by their form and their cause, than by the etymology of the terms, or their proximate constitution. Rokitansky defines exostosis as a purely bony mass, set upon a bone, forming with it an organic whole, and, where it is possible, originating or proceeding from the bone; when its development is complete, and often at the beginning of its growth, its texture is homologous with that of its base and point of origin, whether compact or spongy. The former is the most frequent; and it attains a hardness which has given rise to the term of ivory exostosis; while its colour is generally whiter than that of the bone from which it springs. Of the density of these exostoses, the best proof is that operators are sometimes unable to remove them; in St. George's Hospital Museum we find an exostosis from the orbit which sloughed off on the application of caustic, though Sir Astley Cooper had previously failed in sawing it off; there is another specimen in the same museum, about one inch and a-half in diameter, which took one hour to remove, and more than one saw was spoiled during the time. The exostosis may be entirely sessile, or it resembles a mushroom in its mode of growth, presenting a constriction at its base, which, though it may penetrate deeply, is so fine as to be imperceptible during life.

The surface of these exostoses is smooth, and their outline is commonly a segment of a circle, or of an ellipse their cause is an idiosyncrasy of the individual not referable, as far as we can trace, to any definite constitutional taint. Some of the hard exostoses we meet with are manifestly mere hypertrophies of the normal prominences of the bone upon which they are seated; thus we see the tuberosity of the tibia, the styloid process and similar parts, give rise to these formations.

The bones of the skull are the most ordinary site of hard exostoses; they are also seen in the long bones, and in the pelvis where they may prove an obstacle to parturition. The microscopic appearances are described by Rokitansky as exhibiting a very considerable number of peripheral lamellæ, in which long corpuscles are observed. The Haversian canals are small, and far apart, many of them being surrounded by a distinct and isolated system of lamellæ; large tracts present no corpuscles, while at other spots they are clustered together in dense groups. No other tissue is discoverable in the ivory exostosis.

Fig. 193.



Spongy exostosis on the femur, with a broad base and pointed processes directed downwards; the section shows a cancellous structure, surrounded by a shell of compact bone. The walls of the femur and the medullary cavity, in the situation of the exostosis, are perfectly sound.

(St. Bartholomew's Museum, i. 186.)

Spongy exostoses differ from the compact variety, in being composed of cancelli, containing medullary matter, and surrounded by a shell of bone; they vary much more in size and outline than the former; they spring from the cancellous or compact tissue of the bone, and their surface is continuous with that of the latter. In some cases the medullary cavity of the bone is immediately continuous with that of the exostosis, so that this resembles a diverticulum. In other cases it is separated by a layer of compact bone, as was the case in an instance which was exhibited at the Pathological Society of London,* in 1850, and which was remarkable both on account of its size, and because the base and pedicle were compact, while the remainder was cancellous. The spongy exostosis occurs at all periods of life—when it has attained a certain size it generally remains permanent. Rokitansky describes a process of condensation alternating with one of rarefaction; it is by the latter that he considers the growth of the spongy exostosis outwards to be chiefly affected.

This form of exostosis is sometimes multiple, forming on many bones at once, as if from some constitutional cause. In a case observed by the Editor, spongy growths were found on every bone of the body except the skull, sternum, and the hands and feet; and in most at two spots, *i.e.*, at each extremity of the shaft of the long bones and of the ribs. They were quite unconnected with the epiphysis or with the cartilage.†

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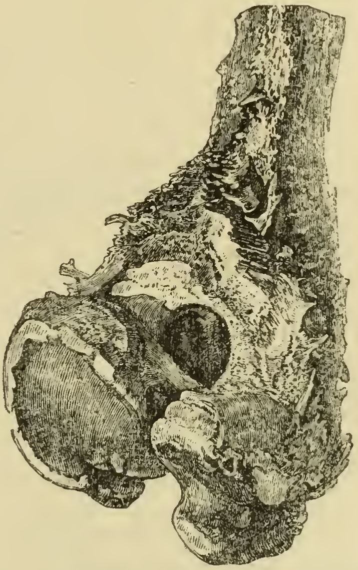
* Report for 1850-51, p. 149.

† "Trans. Path. Soc.," vol. xxiii. p. 208. A similar case in Virchow, "Krankhafte Geschwülste," vol. ii. p. 83.

OSTEOPHYTES.

The osteophyte was first characterized by Lobstein as a bony vegetation which grows from the surface of the bone, or encircles the articulations, and offers the most varied forms. It is distinguished by Förster from exostosis as being a superficial rough, lamellated, or splintery growth, extending over a considerable area, and differing in texture from the adjacent bone, and not continuous with it, but appearing rather as if cemented to it by subsequent growth. The osteophyte chiefly affects the more vascular portions of bones, as their articular ends, their rough lines, or, in the skull, the sutural cartilages; because, as Rokitansky remarks, it is generally the product of an inflammatory process in the superficial part of the bone, and in the periosteum; and hence it is very commonly found adjoining and surrounding not only portions which are inflamed, carious, or necrosed, but also spots of bone affected with various other diseases, which in some stage of their existence have occasioned a reaction in the tissue of the bone. Thus we may refer the osteophyte, in an individual case, to simple inflammation, to rheumatic or gouty inflammation, to syphilis, to new growths, or other causes.

FIG. 194.



Osteophytes, occupying the lower end of the femur. The whole exterior of the bone is roughened by the growth of irregular plates and pointed processes of osseous substance. A large canal, formed by ulceration, passes through the bone just above the condyles; around the lower part of each condyle is a broad rim of new bone. From a man, aged thirty-five, with long-standing disease of the bone.

(St. Bartholomew's Museum, i. 201.)

The diffused and fibro-reticular osteophyte of Lobstein, or what Rokitansky terms the velvety villous osteophyte, forms an osseous layer investing a bone that is otherwise healthy, sometimes removable, sometimes firmly soldered to it; it commonly presents the colour of the bone, or it may be discoloured; by a lens it is found to present a furrowed surface, or to be composed of minute upright spiculæ; the small channels which separate the osseous ridges being in the direction of the vessels of the periosteum. This variety is a very common accompaniment of inflammatory affections of the bone—it is the one which Rokitansky has observed to occur in the skull in females dying shortly after parturition, and which he has therefore called the puerperal osteophyte. The layer of new bone, he says, varies in thickness from a very thin film to half a line, and more; generally occupies the frontal and parietal bones, but is sometimes found covering the whole inner surface of the cranial vault, and scattered in patches over the base of the skull. But the connection of this growth with the puerperal state is very doubtful.

Rokitansky's second variety of osteophyte is the splintered or laminated form, presenting itself in excrescences and lamellæ several lines in length, of a conical shape, and terminating in a sharp point, which are found chiefly in the neighbourhood of the cancellous parts of bone affected with caries.

The next form of osteophyte which we have to consider is that which appears to be mainly the result of chronic rheumatic arthritis; it is distinguished by forming excrescences of a warty and stalactitic character, which are developed in the vicinity of joints of persons labouring under this disease; the articular surfaces may be partially absorbed and present patches of enamel-like deposit, while the new osseous formations are thrown out, as it were, to support the defective mechanism. The osteophyte produced under such circumstances sometimes surrounds the joint and gives rise to a bony ankylosis. The bodies of the vertebræ are frequently found united to one another by osseous vegetations, extending over two or more bones, like bridges; they are analogous to the callus uniting fractures, but appear to be the result of some constitutional cause. Similar bony ridges are also observed to form between the ribs.

The cauliflower osteophyte is described by Lobstein as a large sessile tumour, which is more or less compact at the base, and becomes spongy towards the surface, sometimes attaining the size of the head of a seven-months' child. It may arise from periosteal bony growth, or occasionally merely forms an ossification of the stroma or fibrous envelope of some form of morbid growth, as scirrhous, fibrous, fungous tumours, and the like. Lobstein's general theory of osteophytic growths is, that they consist mainly of an ossification of the tissues surrounding the bone; according to this view, the diffused osteophyte is nothing but ossification of the cellular tissue, uniting the periosteum to the bone; the fibro-reticular osteophyte an ossification of the periosteum itself, the flat and styloid form the ossified tendinous and aponeurotic fibres, while the variety now spoken of and that causing ankylosis is attributed to ossification of the intermuscular cellular tissue; he denies the inflammatory origin of the malady, and sets it down to a morbid hypertrophy.

ENCHONDROMA.

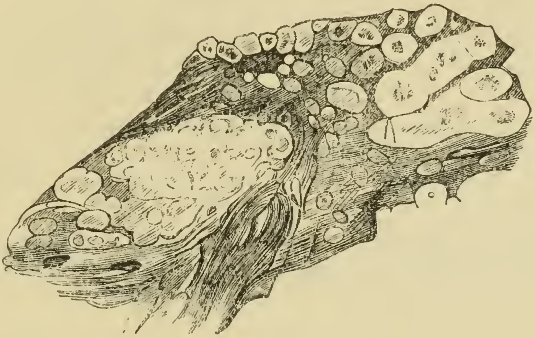
The abnormal cartilage formed in connection with bone or enchondroma consists essentially of the same chemical and microscopic elements, as true cartilage. It occurs more frequently in bone than in any other physiological tissue of the body; the bones of the fingers and toes being chiefly liable, though the ribs, vertebræ, and sternum are not exempt; and cases are recorded where the skull, the ilium, and the long bones have been attacked.

The characters of enchondroma have already been described (p. 141).

The enchondroma is nearly always radically cured by amputation, but rarely gives rise to secondary growths.

The tumour may originate within the substance or on the surface of the bone; the former being called central, the latter peripheral, enchondroma. The rapidity and extent of its growth varies, but generally it is of slow progress, and does not exceed the size of an orange. When seated within the bone, the latter gradually expands with the development of the tumour, yet it is unaccompanied by pain or disorganization of the adjoining parts; when external to the bone it exhibits a lobulated arrangement, and is surrounded by a fibrous sheath.

FIG. 195.



Enchondroma. Portion of the tumour removed from the lumbar vertebræ of a soldier, consisting of nodules of cartilage of various forms, with the microscopic features of fœtal cartilage. In the centre of some of the nodules there are small portions of cancellous bone; the centres of others are softened.

(St. Bartholomew's Museum, xiv. 2.)

The central variety is met with chiefly in the bones of the hands and feet; it presents a semi-elastic feel, and, on section, the knife passes through a thin crackling shell of bone, and then exhibits a white cartilaginous mass, which is occasionally found to contain some small cells, while in some tumours there is an interlacement of fibrous tissue, in which the cartilage is imbedded, thus approximating to fibro-cartilage. They may be solitary, or occur in large numbers in the same individual. A remarkable instance is recorded, in the "Reports of the Pathological Society of London,"* of a boy, in whom the slightest blow produced tumours of this kind. At the time of observation he presented fifteen or sixteen of these swellings, on the fingers and metacarpal bones, one of which had attained the size of an orange, and required removal, solely on account of its bulk. They are liable to ossification, and sometimes ulcerate. Multiple central enchondromata usually occur in children, sometimes with an hereditary tendency.

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* 1848-49, p. 113.

The peripheral enchondroma may occur at any age, and in various parts of the skeleton; sometimes spontaneously, sometimes apparently resulting from injury. It develops from the periosteum or subperiosteal layer of bone, and grows outwards, pushing aside the soft parts. Ossification occurs in these tumours, but not usually to any great extent. They frequently soften or form cysts. They are sometimes combined with myxomatous, and sometimes with sarcomatous structure. In the latter case they are liable to have malignant properties. Sometimes they grow out in a villous or papillary form.

A remarkable case of metastasis of cartilaginous growth is reported by Biesiadecki.* A cartilaginous tumour of the iliac bone, distinguished by a remarkable villous character, gave rise to cartilaginous masses (or cartilaginous thrombi) in the iliac veins; from which were detached cartilaginous emboli. The latter, lodging in branches of the pulmonary artery, gave rise to secondary tumours in the lungs. A somewhat similar case was observed by O. Weber.†

Fatty tumours have, in exceptional cases, been observed in bone. Myxoma is more common, being sometimes combined with enchondroma.

BLOOD TUMOURS IN BONE.

Hœmatoma.—Effusion of blood, either into the subperiosteal tissue or into the medulla, gives rise to the form of tumour, or deposit called hæmatoma. It is usually produced by injury. In the substance of the bone, the degenerated clot may give rise to a blood cyst.

Cephalhœmatoma.—A form of sanguineous tumour of the head, met with in infancy as a result of the pressure exerted upon the cranial bones during parturition, and known by the term cephalhæmatoma, has given rise to much discussion, as the symptoms have been variously explained by different observers.‡

It consists of an effusion of blood between the pericranium and the bone, and is most commonly met with on one of the parietal bones. Rare cases are recorded of an internal cephalhæmatoma, in which the extravasation took place between the dura mater and the bone. All authors are agreed that external cephalhæmatoma occurs most frequently on the right side. Bednar (quoted by Dr. Willshire) found, that of seventy-four examples, forty were on the right, twenty-two on the left, six over each parietal bone, four on the occipital, one over both parietals and the occipital, and one over the frontal bone, the latter being the smallest.

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* "Wiener, Akadem. Sitzungs-berichte," Abth ii. vol. lvii. May, 1868.

† "Virch. Archiv.," vol. xxxv. p. 503.

‡ A good resumé of their opinions is given by Dr. Willshire in the tenth volume of the "British and Foreign Med.-Chir. Review," July, 1852, p. 6.

Older writers have regarded it as depending upon an essential disease of the bone, owing to the hard ring bounding the tumour, inducing the impression that the bone has been excavated. This appears, however, to be strictly a secondary production, the result of an ossifying process on the surface of the bone, and on the surface of the detached periosteum. A further growth, according to Rokitansky, commences at the margin of the denuded part, and produces the deposit of bony matter in the form of a velvety or finely filamentous osteophyte.

The clot passes through the process of softening and absorption common in thrombi. In unhealthy cases, suppuration or puriform softening takes place, which may give rise to extensive suppuration and necrosis of the bone.

VASCULAR TUMOURS.

Tumours of a vascular character are occasionally met with in bone, resembling those composed of erectile tissue, or rather of a congeries of blood-vessels, in soft parts. Mr. Stanley describes a tumour of this kind that fell under his observation, as bearing a close resemblance to certain nævi consisting of dilated blood-vessels, with a fibrous tissue occupying their interstices; hence in a section the tumour presented a cribriform appearance, the orifices being apparently those of divided blood-vessels. This tumour must not be confounded with those enlargements of the bone which are produced by an accumulation of blood, owing to rupture of the blood-vessels. and consequent hæmorrhage into the cancelli, or between the periosteum and the bone. Cruveilhier gives delineations * of a remarkable case, in which a lady, aged thirty-eight, of a good constitution, and without hereditary taint, the mother of eight children, presented a dozen tumours of the size of a walnut, situated on the head. They were soft, and pulsated; the beats were isochronous with that of the pulse, and were accompanied with a blowing noise, similar to one heard at the aorta. There were several similar tumours in other parts of the body, but those of the head only proved after death to belong to the bones. They exhibited on section a filamentous areolar structure filled with blood; the destruction of the bone penetrated to the dura mater; the absorption of the osseous tissue resembled that produced by aneurism; on the external surface there was

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* "Anatomie Pathologique," tom. ii. line xxxiii. pl. iv.

evidence of an attempt at repair, in the shape of osseous vegetations. Cruveilhier is of opinion that in this case the vascular development took place mainly at the expense of the arterial system; he thence infers a general law that there are two kinds of erectile tumour, one of a venous, the other of an arterial character.

Pulsatile and highly vascular tumours of several species beside cancer may give rise to such symptoms as those described. There seem, however, to be real tumours composed of vessels only, true *angiomata*, which have given occasion to the designation "aneurism of bone." *

FIBROUS AND SARCOMATOUS TUMOURS.

Fibrous, or fibroid tumours, sometimes occur centrally, like enchondroma, distending the bone in a similar manner, and sometimes reaching a great size. They may also grow from the periosteum, and then may become partially ossified from the base, or isolated masses of bone (parosteal tumours) may form in them.

Sarcoma of Bone.—The class of bone tumours having the structure of sarcoma is at the present moment very difficult to limit, since the anatomical definition of sarcoma includes most of the tumours hitherto described as cancer of bone.

Spindle-celled sarcoma, or recurrent fibroid, occurs not unfrequently as a growth starting from the periosteum. These tumours are the type of what Sir J. Paget calls recurrent fibroid; their clinical history is that of this form of sarcoma in general (p. 168). They often have a pseudo-fibrous appearance, called fasciculate, and hence have been called fasciculated sarcoma (or carcinoma). They may form bone, or become simply encrusted with calcareous salts. The same form of tumour may start centrally, as in the specimen figured on page 146, in which Virchow was of opinion that there was a combination with cancer.†

Central sarcomatous tumours are mostly of the *myeloid* type, already described, which English surgeons distinguish more clearly from other forms of sarcoma than do the Germans. They are, however, sometimes of fasciculated appearance, and contain spindle-celled structure. The general description of these tumours has already been given. It only remains to say here that when growing from bones they start, in the majority of cases, from the central, *i.e.*, medullary or cancellous part of the bone, but not always; and that tumours containing abundant myeloid cells are found among the ossifying sarcomata of peripheral growth.

Pigmented, or melanotic sarcoma, occurs as a secondary growth.

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* See a case in "Trans. Path. Soc.," vol. xix. p. 349.

† Besides the cases in Paget's "Pathology," very numerous instances are recorded in recent volumes of the "Trans. Path. Soc.," and in Virchow's work on Tumours. See also Arnold, "Virch. Archiv.," vol. lvii.

OSTEOID TUMOUR.

The "osteoid tumour" of Müller has been thought to constitute a transition between the simple bony tumours and the malignant affections of bone. Müller describes it thus:—The osteoid tumour is irregularly lobulated, and is developed with more or less rapidity from the surface of a bone, and consists mainly of osseous tissue, in the interstices of which a non-ossified substance is found of the consistency of fibro-cartilage, which also forms the covering of these growths; the bony matter is more or less porous, and presents all the characters of true bone, while the other constituent offers a greyish white colour, is somewhat vascular, and of firm consistency and difficult to tear. The microscope displays in it a dense fibrous network, with minute interstices containing but few cells and nuclei; it is not cartilaginous in structure or chemical composition, containing neither gelatine nor chondrine.

The tumours thus described are doubtless in part ossifying sarcomata of different species, but one distinct and characteristic form of tissue is found in some of them; that is the osteoid tissue of Virchow, which is more like an imperfect form of cartilage than bone or sarcomatous tissue. Tumours composed of this substance are very rare; we must refer to Virchow* and to the works of Rindfleisch, and Cornil and Ranvier for fuller accounts. These growths are among the tumours called osteoid cancers by Sir J. Paget.

CANCER OF BONE.

The occurrence of primary cancer of bone is, whatever definition of cancer we adopt, rare; but taking it in the sense formerly defined, rarer even than the descriptions of surgeons would lead one to suppose, since most of the tumours thus named from clinical characters would, according to the definition here adopted, come under the head of sarcoma, especially of the round-celled or medullary form. As it is impossible, in the standard descriptions of bone cancer, to separate the carcinomatous from the sarcomatous forms, the general assertions made respecting the growth and other features of cancer must be taken to apply equally both to soft rapidly-growing forms of sarcoma, and to medullary carcinoma in the strict sense.

Epithelial Origin of Cancer.—Another difficulty must be faced before we can admit the existence of true primary carcinoma of bone. In most of the recent German publications on morbid growths, it will be found that the name cancer is confined to tumours originating in glandular or superficial epithelium, and of which the cells (contained in the alveoli) have a more or less epithelial character. This excludes from the class of cancers nearly all the malignant tumours arising from bone, and, accordingly, Billroth has proposed for some undoubtedly alveolar tumours, which are anatomically cancer, in the sense in which the word is used in this book, the name alveolar sarcoma. This is understood as differing from cancer only in the fact that the cells have not an "epithelial" character. Whether the name carcinoma or sarcoma should be given to such tumours, depends on a variety of considerations which cannot be discussed here. We only wish to point out that "alveolar sarcoma" is in many cases synonymous with carcinoma, in the sense in which we use the word. Finally, it must be stated that we do, from time to time, meet with tumours growing from bone which are both "alveolar" and "epithelial," and to which no pathologist would refuse the name of cancer, either on anatomical or clinical grounds. Such cases are explained, by those who deny the origin of cancer from anything else than epithelium, in two ways: first, they say the occurrence of such growths is so rare and exceptional as to be altogether unimportant; secondly, they suppose that there may have been, in such cases, some inclusion of epithelial or epidermic structure within the bone in the course of development; as appears to be the case in the dermoid cysts, found in exceptional situations, formerly referred to. This explanation appears forced, and we can only say that the "epithelial" theory of the origin of cancer does not at present appear susceptible of application to all cases, and must therefore be regarded as, up till now, insufficiently established. The definition of Virchow forms the best practical basis for anatomical classification.

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* "Krankh. Geschwülste," vol. ii. p. 292, et seq.

Primary Cancer of Bone.—Several varieties have been seen. Epithelioma is recorded as having occurred in two cases by Förster.* Virchow and Weber have seen similar cases.† Messrs. Cock and Bryant describe another.‡ In such cases, the epithelial character of the cells could admit of no mistake.

The ordinary medullary carcinoma is what is sometimes called alveolar sarcoma. Cases are not very uncommon. They occur chiefly in certain situations, such as the lower end of the femur or humerus, the cranial bones and the vertebral column, sometimes in other situations, as the tibia or pelvic bones. A perfect instance of this form of tumour is one first described as osteo-sarcoma, but which, in its further development, and especially in the lymphatic and secondary tumours to which it gave rise (as we have, since the publication of the case, had an opportunity of observing), was precisely what has been generally understood by medullary carcinoma,§ or lately, alveolar sarcoma. These tumours are sometimes found to develop in a remarkable manner, apparently simultaneously from many parts of the skeleton.

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* "Handbuch der Path. Anat.," vol. ii. p. 895.
† Lücke, "Geschwülste," p. 207.
‡ "Lancet," vol. i. 1859.
§ Croft, "Trans. Path. Soc.," vol. xxiii. p. 203.

Scirrhus has never been observed as a primary growth in bone.

Colloid is almost unknown primarily affecting the bones. Great interest, therefore, attaches to the case reported by Dr. Moxon ("Trans. Path. Soc.," vol. xxii. p. 206), in which this form of cancer affected simultaneously a large number of bones. The growth was strictly alveolar, and the alveoli contained colloid matter, with a few cells. If not called cancer, it could only receive some such name as alveolar sarcoma with colloid degeneration.

Secondary cancer of the skeleton is far more common, and in this form most kinds of cancer have been found.

Scirrhus is the least frequent, but distinct isolated tumours are sometimes seen. Instances are given by Sir J. Paget, in his "Surgical Pathology." Medullary cancer not unfrequently occurs subsequently to the same affection of the soft parts, and often in many bones at once. It may, like primary encephaloid, appear either in the form of distinct tumours or as an infiltration.

Secondary colloid has rarely been observed in bone. Mr. Stanley records a case affecting the bone of a finger, and another occurring in a rib. Rokitansky relates one in which the right upper maxilla was the seat of the growth, and where the peripheral follicles of the growth were developed into large cysts.

General Characters of Bone Cancer.—The general mode of growth in all soft malignant tumours of bone, whether encephaloid or sarcomatous, is much the same. Separate tumours arise either centrally or peripherally. In the former case they fill the medullary cavity, and expand the external shell of bone, or else infiltrate it, converting it into cancer, and proliferate beyond it. The bony shell sometimes found on the outside of such tumours is, however, of subsequent formation. Bone is often formed in the tumour, and when macerated appears as the curious structure known as spina ventosa. The original bone is perforated, or in great part destroyed. In the infiltrated form of encephaloid, the cancelli and Haversian canals are filled with a reddish, fatty-looking substance, which causes an absorption of the cancellar septa, and thus becomes one of the various morbid conditions to which a great fragility of the bones is attributable. The tissue presents the characters of medullary cancer; it varies in vascularity; at times, either from the large number of small arteries passing through the growth, or from the vicinity of a large artery, it puts on the character of an erectile or aneurismal tumour, and exhibits marked pulsation. It is probable that the majority of cases of aneurism of bone are tumours of this kind. The fragility which was noticed in the individual case before alluded to, is a quality often spoken of by authors as associated with cancer, without determining or assuming the existence of actual carcinomatous disorganization of the bone; it is also observed in deep-seated constitutional affections of a syphilitic, scorbutic, and arthritic character, and cases are recorded of extreme fragility as a mere result of old age; it is not, therefore, inconsistent to assume that, when accompanying carcinomatous affections, it may be sometimes the result of an atrophic state resulting from mal-nutrition, without being necessarily accompanied by any actual carcinomatous disease of the bone.

That bone cancers, or malignant sarcomata, may give rise to secondary formations in other organs, especially in the lungs, follows from the general principles before laid down. Such secondary tumours are sometimes ossified, and sometimes not.

TUBERCLE IN BONE.

The presence of tubercle in bone is an undoubted pathological fact, but its frequency has been over-estimated by some authors, as it has been underrated by others. In the former case the error has arisen from every evidence of osseous disease in scrofulous subjects having been regarded as actually resulting from the deposit of tubercular matter in the bone, and from concrete pus having produced appearances closely resembling those presented by tubercle in a certain stage of decay. We have already pointed out that there is no evidence that such cheesy masses were ever tubercles; but it is fair to say that they have not often been proved not to be so; and it remains to be seen what improved methods of investigation may establish, with regard to the tubercular origin of some of them.

Grey miliary tubercles are very rarely seen; not often even in cases of general tuberculosis, though it must be remembered that the bones are seldom examined in this disease.

According to MM. Cornil and Ranvier, who have made special researches on this subject, miliary tubercles are found in the spongy tissue, more especially of the vertebræ, the sternum, and the ribs. They describe isolated granulations and also confluent granulations; the latter forming a species of infiltration.

SYPHILITIC TUMOURS OF BONE.

Nodes or gummata occur most frequently in the periosteum, that is, in the subperiosteal layer, already spoken of, as the result of a chronic inflammation. They form distinct elastic swellings, sometimes so hard as to simulate bony growth; actual bone is not often found in them, though it has been seen in nodes on the tibia. On cutting into them they are found to be composed of a yellow translucent elastic substance, sometimes yielding a thick gummy fluid, the minute characters of which have already been described. This tissue is particularly liable to decay, through a species of fatty degeneration, leading to the production of a dry yellow, sometimes cheesy mass, which may in the end become completely absorbed. When this happens a depression is usually left in the skull, showing the participation of the bony tissue, which is usual in so-called periosteal affections. Actual suppuration is rare. Sometimes a more fibrous mass results, which is permanent for a long time; or, on the other hand, ulceration may ensue; though this will not be formidable unless the central part of the bone be also affected. This combination does, however, sometimes present itself, and syphilitic ostitis, or osteomyelitis, may accompany the periosteal affection; as they may also occur independently. The medulla, and especially that portion of it contained in the spongy bone and diploe, is here the seat of the morbid process. This may, as in its more superficial seat, produce gummatous tumours, which undergo caseation, while the bony tissue included within them perishes (by necrosis), and various processes of demarcation, as suppuration, granulation, or osteosclerosis are set up around. The tumours thus produced are, however, not considerable.

Syphilitic caries and necrosis are properly treated under this head, since they are not, as Mr. Holmes well remarks, primary affections of the bones, but the result and termination of nodes, or of inflammation of the bone, or of ulceration in the soft parts around.

Syphilitic ulceration of bone occurs in two forms. In the first, or *annular*, a small carious spot appears first, and a wide ring of bone surrounding this is ultimately separated by a sequestrating furrow, so that a depression much larger than the original spot is formed. This is a combination of caries with necrosis. The "tubercular" ulceration begins with syphilitic inflammation of the compact tissue, which causes an elevation on the surface. The inflamed bone soon becomes dotted over with numerous little pits, or depressions, which coalesce, and form ulcers, usually oval or round, penetrating deeply into the interior of the bone. By the confluence and extension of these forms, especially the annular, complicated or reticulated forms are produced. The severer forms of caries occur in cachectic constitutions, or, as is thought, when much mercury has been taken. They are less frequent now than formerly.

PARASITES.

Hydatid or echinococcus cysts occur very rarely in bone. A very remarkable instance is described by Mr. Keate,* in which a large tumour in the frontal bone of a young woman, aged eighteen, was formed by the development of hydatids between the plates of the bone. Frequent attempts to destroy the cyst and its contents, by escharotics, after removal of a portion of the external osseous sheath, failed; and the constant sprouting out of fresh hydatids at last induced Mr. Keate to saw off the entire tumour, after which the girl completely recovered. The diameters of the exposed surface were $4\frac{1}{2}$ by 4 inches. In the very compact and hard bony substance, forming the base of the tumour, were five or six cells, containing hydatid cysts. It appears that from these cells the hydatids were constantly regenerated, forcing their way into the large cavity of the tumour, and yielding to no treatment, as the remedies failed to destroy the matrix from which they pullulated.

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* "Med.-Chir. Trans.," vol. x. p. 278.

Mr. Stanley quotes some cases from his own experience, and that of other observers, from which it appears that the bones of the skeleton are all equally liable to be attacked. The development of hydatids induces a gradual, painless expansion of the bone, which may thus become perforated, and allow of an escape of the hydatids. The cavity of bone in which the hydatids form is lined by an adventitious cyst, and this is said to be liable to excite inflammation in the surrounding bone, as well as to induce purulent products in the cysts. Cysts of this description often contain matter, to the naked eye, absolutely identical with laudable pus, which, however, under the microscope, exhibits none of the characters of pus.

Cysticercus cellulosæ was observed in one case by Froriep in the first phalanx of the middle finger, where it had developed in the midst of the bone, and by secondary inflammation destroyed the external wall on the volar side.

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